UNIVERSITY OF ALBERTA

Financial & Health Costs of Pesticide Use in Growing Conventional and Genetically Modified Potatoes in Prince Edward Island

by

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ABSTRACT

This study focuses on economic analysis of the use of conventional and genetically modified potatoes in Prince Edward Island in the context of health risks of air-borne pesticide residues and consequent potential impacts on the health of farmers and their families. Estimates of health impacts and other related costs are developed for conventional potato growing practices and in growing genetically engineered potatoes (NewLeaf, NewLeaf Plus and NewLeaf Pro potatoes, each genetically modified for particular traits). From interviews with potato specialists and potato farmers, it was discerned that pesticide applications were reduced when using the genetically modified potatoes. The extent of reduction in pesticide applications is documented and translated into financial benefits and potential health benefits received by the pesticide applicator. It is concluded that the financial benefits gained from the use of fewer inputs are much more substantial than are the health costs associated with the exposure to pesticides.

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CHAPTER 1: INTRODUCTION

1.1 PRINCE EDWARD ISLAND AGRICULTURE OVERVIEW

Prince Edward Island (PEI) constitutes a very small area on the east coast of Canada, approximately 560,000 hectares, and is responsible for producing almost one third of the potato crop in Canada. Potatoes are PEI's primary cash crop, capturing one third of total cash farm receipts of the province.

Farmers in PEI grow potatoes for three specific market segments: seed, table potatoes and processing. Seed potatoes are sold to commercial growers and home gardeners for the next year's crop; table potatoes are sold in the retail and food service sectors; and processing potatoes are manufactured into French fries, potato chips, and other processed potato products [PEI Potato Board 2002].

Almost 50% of the potatoes grown on PEI are used by the processing industry. Most are processed into frozen potato products, which are available across Canada and also exported to approximately 30 countries around the world. The two main French fry processors in the province are Cavendish Farms (a division of Irving Canada) and McCain Foods. After processing, French fry potatoes from PEI are available across Canada as well as exported [PEI Potato Board 2002].

In addition to sales in the Canadian market, seed and table potatoes are shipped to over 20 countries each year. Recent customers are in countries such as Venezuela, Italy, Ukraine, Portugal and Thailand. While markets vary from year to year, over the past few years about 50% of PEI table potatoes have been sold in Canada, 20% have been

marketed in the United States and the remaining 30% are sold to other countries [PEI Potato Board 2002].

Potatoes are the fourth largest food crop in the world, popular partially because of their nutritional value. However, with its popularity comes the need to have good crop yields in succession.

A number of pests and viruses plague PEI potato crops. The most prominent pests include the buckthorn aphid, Colorado potato beetle, European corn borer, green peach aphid, nematodes, potato aphid and potato flea beetle. The most common viruses/diseases in PEI potatoes are early blight, late blight, common scab, dry rot, potato leafroll virus (PLRV), gray mould and bacterial ring rot. Late blight requires the largest number of sprays of chemicals during the growing season on PEI. To fight these pests and viruses/diseases, farmers use crop rotations, integrated pest management (IPM), insecticides/fungicides and for a few years, potatoes that had been genetically modified to repel pests. Table 1.1 gives a brief overview of the history of potato production in PEI and illustrates when genetically modified potatoes came into the picture.

Table 1.1 - Timeline of genetically modified potatoes on PEI

Date	Research	Farmer/Producer	Trade/Industry
1771		First record of potatoes grown in PEI[a]	
1790			Exporting began[a]
1992			Potato receipts equal to 32% of total farm cash receipts[b]
1994	First fish kills reported[c]		

1995	Health Canada and CFIA approved NewLeaf potato for growth and consumption[d]	
April 1998	Announcement that NewLeaf Pro will be available in 2002 - resistant to Colorado potato beetle, potato leafroll virus, potato virus Y and late blight[e]	
1998	Approval for NewLeaf Plus (US) - resistant to Colorado potato beetle and potato leafroll virus	
1999	Approval for NewLeaf Plus in Canada[d]	
1999	Approval for NewLeaf Y (Canada & US) - resistant to Colorado potato beetle and potato virus Y[d]	
Summer 1999	8 fish kills linked to pesticide runoff[c]	
1999		Potato receipts equal to 55% of total farm cash receipts[b]
Nov 1999		McCains announced they would not accept GM potatoes for the 2000 harvest year[f]
21 Mar 2001		Monsanto dropped NewLeaf potato from US and Canadian markets[g]

[a] Boswall 1998.
[b] Government of Prince Edward Island 2002.
[c] Freeze 2002.
[d] Health Canada Food Directorate. www.hc-sc.gov.ca
[e] Potato Blossom Industries Inc. 1998.
[f] Byfield 1999.
[g] Athayde 2001.

1.2 GENETICALLY MODIFIED POTATOES

The Royal Society of Britain concluded from data gathered from 1998 to 2001 that existing genetically modified crops pose no more risk to human health than do conventional foods [The Economist 2002]. The biotechnology "revolution" has produced a number of genetically modified foods that have been engineered to be resistant to particular diseases or pests. The hope for this application of biotechnology has been to reduce the number of pesticides used in agriculture while ensuring that the expanding world population will have a continued source of food.

The earliest form of approved genetically modified potatoes were available to farmers from Monsanto in 1995, with the first growing season in 1996. These first modified potatoes were called NewLeaf potatoes, a Russet Burbank cultivar that was modified for resistance against the Colorado potato beetle. The second variety of genetically modified potatoes was called NewLeaf Plus, a Russet Burbank cultivar that was resistant to the Colorado potato beetle and the potato leafroll virus. As Table 1.2 illustrates, both of these varieties were grown on PEI until 1999 [MacPhail 2003, Health Canada 1999a, Health Canada 1999b].

Table 1.2 - Genetically Modified Potatoes: Traits and Stage of Development

Genetically Modified Potato	Traits	Stage of Development[a]
NewLeaf (Russet Burbank)	resistant to Colorado potato beetles	approved for growth and consumption in Canada in late 1995
NewLeaf Plus (Russet Burbank)	resistant to Colorado potato beetles and potato leafroll virus	approved for growth and consumption in Canada in 1999
NewLeaf Y (Russet Burbank)	resistant to Colorado potato beetles and potato virus Y	approved for consumption in Canada in 1999, but were never grown
NewLeaf Pro	resistant to Colorado potato beetles, potato leafroll virus, potato virus Y, and late blight	developmental stages - was to have been ready for growth by 2002

[a] Sources: Health Canada 1999a, Health Canada 1999b, Health Canada Food Directorate

Additional cultivars, including Atlantic, Superior, and Shepody, were reported as being in the process of modification to have the same traits as the NewLeaf Russet Burbank potatoes [NatureMark 2004].

1.3 OVERVIEW OF MODIFICATIONS

The NewLeaf Russet Burbank potato included in its genetic structure two new genes isolated from naturally occurring bacteria. These genes produce two proteins, the first derived from the bacteria *Bacillus thuringiensis* (Bt), which protects against the Colorado potato beetle, while the second is a biological marker that allows researchers to identify the modified potatoes.

The NewLeaf potato cannot be distinguished from other potatoes by appearance,

taste, nutritional content or cooking quality. It costs less to grow because it has incorporated the Bt insecticide into its genetic makeup. This means that farmers will apply fewer insecticides to their fields to control the spread of the Colorado potato beetle [Arsenault 2002]. The other NewLeaf variety potatoes were developed based on these principles, adding the additional genes that provided resistance to potato leafroll virus and potato virus Y.

In November 1999, McCain Foods, a world-scale food processor, sent producers a letter advising farmers that they would not accept genetically modified potatoes for processing in the 2000 harvest year. This move was said to be dictated by market forces for McCain's products. This initiative is understood to have largely resulted from decisions made by major representatives of the fast food industry, who notified McCain's and Cavendish Farms that they would no longer purchase French fries made with genetically modified potatoes due to consumer preferences.

1.4 IMPLICATIONS OF THE GENETIC MODIFICATION OF POTATOES

Potato leafroll virus infected potatoes develop lesions, a dark brown tissue that has to be removed before French fry processing. A leafroll resistant potato would reduce the incidence of this virus, as well as reducing the cost of battling aphids, which carry the virus [Alberta Report 1998]. Since the majority of French fries are made from the Russet Burbank potato, the impact of the ability to continue to use genetically modified potatoes would have been appreciably lower insecticide costs. The genetically modified varieties were demonstrated to have similar yield, after correcting for specific PEI growing

conditions, to the conventional varieties, but required fewer pesticide applications [MacPhail 2003].

Data yielded by field trials in PEI have shown that GM potatoes have both cost and environmental benefits. For example, if the NewLeaf Pro potato that was in the process of being genetically altered for Colorado potato beetle, potato leafroll virus, potato virus Y and late blight resistance had been available and used in fields, the number of pesticide applications would be reduced by approximately 75% [Arsenault 2002]. Consequently, contamination by pesticides carried to rivers by soil erosion and runoff would have been greatly reduced, resulting in fewer fish kills [Arsenault 2002].

1.5 OUTLINE OF PROBLEM AND APPROACH

Pesticides contain active ingredients, as well as inert substances, that are potentially dangerous to human health and the environment, as well as being costly farm inputs. Exposure to some of these active ingredients can increase the risk of contracting cancer, respiratory problems, and other health problems. Genetically modified potatoes are one way to potentially reduce the exposure of the population of PEI to these chemicals. An illustration of the damage function approach that will be used in this study is given in Figure 1.1:

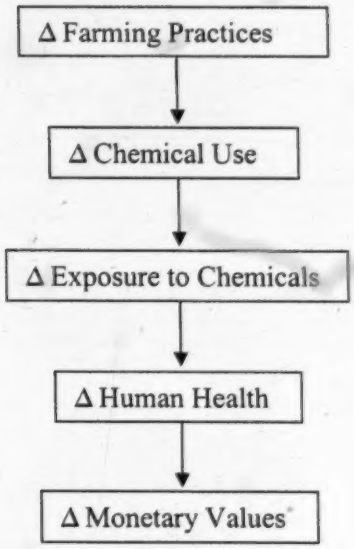

Figure 1.1 Illustration of the steps taken in the damage function approach

Figure 1.1 shows the steps that have been taken in this study, from the change in farming practices when moving from growing conventional potatoes to growing genetically modified potatoes, to the effect that this change will have on chemical use. The change in chemical use is translated into the change in the exposure to chemicals. This leads to a change in human health and the consequent estimation of monetary values associated with health costs and chemical input costs.

Through the use of partial budgeting techniques, the potential savings to farmers are estimated on a per hectare basis, by comparing farming practices using conventional and genetically modified potatoes. These calculations of savings are based on the cost of pesticides, cost of seed, and the cost of contract rates for applying the chemicals.

To estimate the impact on farmers and their families associated with lower use of pesticides, quantitative estimates of carcinogenic risk from inhalation exposure for three identified chemicals are applied for the relevant farmer population of PEI. The health benefits of the potential reduction in exposure when fewer chemicals are used are then estimated in monetary terms, by conversion into monetary values using the Value of a Statistical Life approach.

Finally, discussion emphasizes that this approach is only one step in a more extensive assessment that would consider total benefits and costs associated with using genetically modified potato seed. The potential environmental benefits from reduced pesticide use are not explored in depth in this paper.

1.6 STUDY PLAN

This thesis is organized as follows: The next chapter provides a detailed literature review of cost benefit analysis and how the concept of the value of a statistical life is developed. Chapter 3 provides a discussion of potato cultivation methods and pesticide chemical families. In Chapter 4 the development of the partial budget and the results are discussed. Chapter 5 focuses on the dose response function model development. In Chapter 6 the results from the previous chapter are presented and discussed. Chapter 7 presents a summary and a brief discussion of the limitations and conclusions of the study.

CHAPTER 2: LITERATURE REVIEW

2.1 DAMAGE FUNCTION APPROACH

Damage functions usually take the form of translating the subject being studied (in this case, the active ingredient in a pesticide being released into the air) into a quantifiable measurement and then into measures of damage done to human health, materials, plants and animals, visibility and aesthetics, and ecology by using a dose-response relationship. These effects are further translated into economic values, with externalities being identified [Matthews 2001]. Matthews (2001) states that in practice, mortality losses dominate the valuation. Damage function approaches can use the techniques of benefit-cost analysis and the concepts of the cost of illness and value of a statistical life to quantify effects.

2.1.1 Cost Benefit Analysis Technique

This approach focuses on measures of the costs and benefits of implementing a policy or project. In general, if the social costs exceed the benefits, then the policy or project is inefficient and should not be undertaken.

The first step necessary when attempting a benefits-cost analysis is to determine the net present value of all the social costs (PVC) and benefits (PVB) of the proposal.

$$PVC = C_0 + \frac{C_1}{(1+r)} + \frac{C_2}{(1+r)^2} + \ldots + \frac{C_T}{(1+r)^T} = \sum_{t=0}^{T} \frac{C_t}{(1+r)^t}$$

where C_t is all of the proposal related costs in year t, T is the life of the proposal in years, and r is the rate of discount, which reflects time preference (based on the feature that a

dollar today is worth more than that same dollar one year from now). These costs represent the costs encountered that are over and above those that would be encountered in the absence of the proposal. Similarly,

$$PVB = B_0 + \frac{B_1}{(1+r)} + \frac{B_2}{(1+r)^2} + \ldots + \frac{B_T}{(1+r)^T} = \sum_{t=0}^{T} \frac{B_t}{(1+r)^t}$$

where B_t is all of the benefits resulting from the proposal in year t. Benefits are defined as the difference between benefits that are present with the proposal as opposed to without it.

Typically, the next step in the analysis is to determine the difference between PVC and PVB, that is, the net present value (NPV):

NPV = PVB - PVC.

If NPV>0, the proposal adds to the welfare of society and is deemed to be economically efficient. If NPV<0, the present value of costs is greater than the present value of benefits and the proposal should not be undertaken because society will be a net loser.

2.1.2 Cost of Illness Technique

As stated in Stratus Consulting (2001), there are a number of potential economic and social costs associated with adverse health effects resulting from air pollution. These costs can be transferred to other diseases resulting from other sources. They include:

1. Medical Costs: out of pocket expenses for the affected individual/family, costs paid by public health care, private insurance, and so on;

2. Work loss: monetary value of lost productivity (lost income whether the affected individual is compensated for time off due to illness or not);
3. Increased costs for chores and care giving: special care giving and services that are not reflected in medical costs;
4. Other social and economic costs: restriction on/reduced enjoyment of leisure time, pain and suffering, anxiety about the future, concern and inconvenience to family members and others.

Cost of illness (COI) measures only take into consideration medical costs and lost income as a proxy for work loss and therefore do not take into account the total welfare impact of an adverse health effect. They are, in general, not consistent with economic welfare theory.

2.1.3 Monetary Valuation of Nonfatal Cancers – Cost of Illness Approach

Willingness to pay (WTP) measures for nonfatal cancers are not available, therefore the cost of illness (COI) approach is the most commonly used. The best-suited COI measure is "an incidence-based measure of the present value of the stream of costs a patient can expect to incur over the course of the illness [Stratus Consulting 2001:5-24]." This COI measure normally includes the expected future health care costs, as well as loss of productivity due to illness. The alternative to this measure is a prevalence-based COI measure, which incorporates the health care costs and productivity losses that accrue to all persons with cancer in a given time period. Prevalence-based measures are not very useful for assessing the financial benefits of preventing new cases.

Stratus Consulting (2001) refers to a cancer study performed by Hartunian et.al

(1981) in which costs were estimated in two parts: direct and indirect costs. The direct costs depended on the site and the extent of the cancer while the indirect costs captured the difference in the expected earnings of individuals with and without cancer. Indirect costs depend on a number of factors including the age of the cancer patient, the site and extent of the cancer, sex of the patient and the survivability of the cancer [Stratus Consulting 2001].

The COI approach is considered to give the lower range of values [Stratus Consulting 2001]. This is because WTP measures are not incorporated into the values, therefore, values for avoided pain and suffering and restriction of the individual from non-work activities are not captured in the COI measures.

2.1.4 Monetary Valuation of Fatal Cancers – Value of a Statistical Life Approach[1]

Value of a Statistical Life (VSL) estimates have been developed in a number of studies by using evidence based on market choices involving tradeoffs between risk and money. Governments and their agencies have used these VSL estimates as a reference point for assessing the benefits of risk reduction efforts. VSL estimates are intended to show the benefits gained from risk reduction efforts as the reduced probability of death of the affected individuals and not the value of lives saved ex post. Economic literature focuses mainly on measures of willingness to pay (WTP) or willingness to accept compensation for risk of the cost of a life that are seen in labour markets, and more recently on the basis of information on price risk/price safety tradeoffs in product markets

[1] This section was adapted from Blomquist (2000), except where otherwise noted.

such as for automobiles and fire alarms [Viscusi & Aldy 2002].

The term "Value of Life" refers to the amount of money in dollars that an individual is willing to trade for small changes in their own probability of survival. For example, think of a group of 10000 people who have the same information and identical preferences. Assume it is known within this group that x people in the group will die in the next year. It is also known that if sufficient funds are raised, the group mortality could be reduced to x-1. If it was known that each person would pay $500 to reduce the mortalities by 1 person, then the value of life becomes $500 per person times 10000 people, or $5 million. This value is normally called the value of a statistical life because the identity of the additional survivor is unknown at the beginning of the exercise.

VSL can also be viewed as a standardized value of a change in mortality risk. That is, the reduction in deaths by 1 person in 10,000 means that an individual's probability of death is reduced by 0.0001. For convenience, this is standardized to a unit change of 1, and $500 is multiplied by 10000 (to standardize 0.0001 to 1), giving a value of $5 million.

To carry the above example one step further, consider a simple utility function of an individual's expected utility:

$$E(U) = P\,U(C) \qquad (1)$$

where E(U) is an individual's expected lifetime utility; U is a utility function with marginal utility $U^{1}>0$ and diminishing marginal utility, i.e. $U^{11}<0$; C is consumption and P is probability of survival.

If equation 1 is differentiated with respect to consumption, keeping the individual

at the same level of E(U), solving for dC/dP will yield the rate at which the individual is willing to trade off current consumption for a small change in the probability of survival. That is,

$$dC/dP = - U(C)/PU^{1} \quad (2)$$

Equation 2 indicates that the rate at which an individual is willing to trade consumption for a small reduction of the probability of mortality is equal to the negative of the utility of consumption divided by the expected marginal utility of consumption. Expressing consumption in monetary terms, dC/dP would be equal to the expected monetary value of the utility of consumption, or an individual's willingness to pay for risk reduction.

Several inferences can be made from the equation 2 relative to the value of life. First, the equation shows that the amount an individual is willing to trade for a small change in his or her probability of survival depends on P, which is in the denominator. This means that the value of life can be expected to increase as P decreases.

Second, value of statistical life depends on distinct factors that are reflected in the individual's utility function. The U(C) can vary among individuals with the same level of consumption, due to differences in preferences that may be associated with family situation or environment. For instance, individuals with a close, supportive relationship with family members, individuals who live in a clean environment, or individuals who live in societies with an advanced social framework can be expected to have higher values of statistical life [Blomquist 2000].

Lastly, value of statistical life depends on C. Since C can be expected to increase

as income increases, U(C) and the value of statistical life will be higher for individuals with higher levels of income and consumption.

Questions have been raised concerning how government policy makers should reconcile the broad range of VSL estimates in the economic literature. For purposes of policy should individual's income or age be considered as variables?

Viscusi and Aldy (2002) attempt to answer these questions by using a meta-analysis approach to explore the relationship between income and WTP based on 49 studies of US and International VSLs reported in their paper. They conclude that the income elasticity for a VSL is less than 1.0 and is normally between 0.5 and 0.6, depending on the number of variables used in the analysis. This indicates that the income variable is inelastic and therefore does not factor into VSL estimates as much as one might think. This same type of meta-analysis approach was used by Viscusi and Aldy to assess how age influenced VSL estimates. They conclude that as age increases, VSL decreases, for the reason that you cannot at 25 "save" good health for age 75.

Despite concerns regarding the use of VSL estimates in policy making decisions, this approach is viewed to have fewer limitations than the COI approach, which can only take into account the direct costs of an illness (eg. Hospital bills, income lost while sick, etc).

Typically, VSL estimates that are used in Canada apply Canadian data where available, while US data are used when information is missing. The US data are converted to Canadian dollars by using a purchasing power parity (PPP) index, then the resulting dollar amount is inflated to a specified base year using the Canadian consumer

price index (CPI) [Stratus Consulting 2001].

Stratus Consulting (2001) suggest an "average range" of VSL estimates taken from Canadian and international data, in 1996 dollars, as $3.1 million (low estimate), $5.2 million (central estimate) and $10.4 million (high estimate). These estimates are based on samples of working age adults. Since individuals of retirement age are not well represented in the samples, the reported values apply only to individuals under the age of 65. To overcome this restriction, a study by Jones-Lee (1985) is used by the authors to calculate a weighted average VSL based on the approximate age distribution of the US population age 65 and older. This results in an adjustment to VSL of 75% of VSL estimates for adults under 65. The Stratus Consulting (2001) authors then assume that 85% of particulate-related fatalities are experienced in those 65 and older. The results of these adjustments are given in Table 2.1.

Table 2.1: Selected Monetary Values for Mortality Risks of Air Pollution

Population Group	Selected VSL Estimates (1996 C$ million)		
	Low	Central	High
65 years old	2.3	3.9	7.8
< 65 years old	3.1	5.2	10.4
Age weighted average VSL[a]	2.4	4.1	8.2

[a] Assuming 85% of deaths are individuals aged 65 and over
Source: Stratus Consulting 2001

2.2 MONETARY VALUATION OF CANCER RISKS

Since there are no adequate empirical estimates of WTP for reducing cancer risks and mortalities specifically related to cancer, one approach used has been to combine VSL estimates with adjusted COI estimates for nonfatal cancers based on the average cancer survival rate [Stratus Consulting 2001]. Table 2.2 is based on exposure to three pollutants that result in new cancer cases where the type of cancer associated with the pollutants is not further specified.

Table 2.2: Monetary Values for Cancer Effects (1996 C$)

	Value per Nonfatal Cancer Case	Value per Fatal Cancer Case[a]	Average Value for All Cancer Cases[b]
Low	$153,000	$2.6 million	$1.6 million
Central	$305,000	$4.3 million	$2.7 million
High	$610,000	$8.6 million	$5.4 million

[a] Reflecting that approximately 70% of cancer deaths in Canada are experienced by those over age 65
[b] Based on the average 5 year survival rate of 40% for all cancers in Quebec
Source: Stratus Consulting 2001

2.3 HEALTH RISK ASSESSMENT PROCESSES

Health risk assessment is based on four processes: Hazard Identification, Dose-Response Assessment, Exposure Assessment and Risk Characterization. Figure 2.1 illustrates this concept:

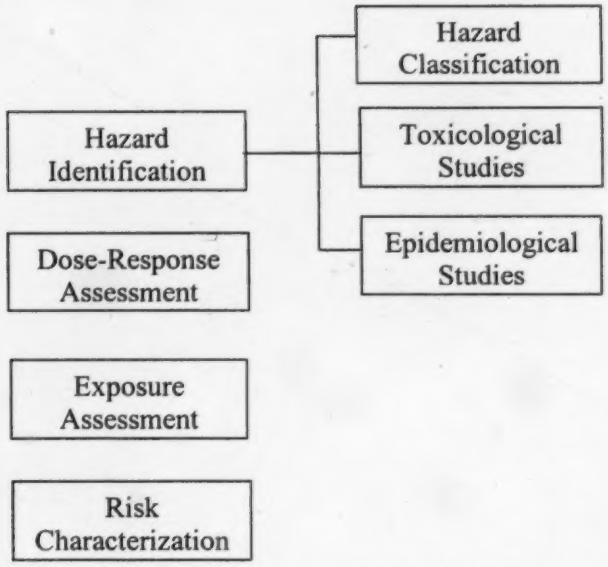

Figure 2.1 Risk Assessment Processes[2]

2.3.1 Hazard Identification

Two pieces of information are needed to determine the potential of an agent to cause adverse health effects: types of injury and conditions of exposure. This information characterizes the 'inherent toxicity' of the substance in question [Reed 1996c].

2.3.1.1 Hazard Classification

Toxicity can be characterized by different biological target organs/systems or outcomes. In risk assessment, hazardous substances can be classified as: carcinogens (ability to produce tumours in specific circumstances); mutagens (capable of causing

[2] Source: Jardine 2004

genetic damage by damaging genes or chromosomes, including DNA damage); neurotoxins (capable of causing effects on the nervous system); developmental/reproductive agents (capable of causing effects on the developing fetus or on reproduction) [Jardine 2004]. Classifications are made on a "weight of evidence" assessment, based on quality of data specifics of the study design, and the overall strengths and limitations of the study. Classification systems have been developed for carcinogens by the International Agency for Research on Cancer (IARC) [IARC 2004], the United States Environmental Protection Agency [USEPA 2004] and under the Canadian Environmental Protection Act [CEPA 2004]. These are based on the availability and certainty of the information linking exposure to the hazard and cancer outcome.

2.3.1.2 Toxicological Studies

Toxicology is the study of the adverse effects of xenobiotics (substances foreign to the body). It has a quantitative basis in the dose-response relationship. Toxicity considers exposures (acute vs. chronic), responses (immediate vs. delayed) and severity (mild, serious, or life-threatening). Toxicity studies are conducted on animals; however it is difficult to relate results from laboratory animals to humans. This is due to the fact that for practical reasons, toxicity testing is done at very high doses so that researchers can see an immediate effect. It is then difficult to relate the results of these tests to the low doses that people are usually exposed to. It is also difficult to relate the outcomes observed in animal species to the expected outcomes in humans [Jardine 2004].

2.3.1.3 Epidemiological Studies

Epidemiological studies focus on the frequency, distribution and cause of disease within a human population. They are considered the best source of information because they are based on human exposures. The best epidemiological studies will give a clear and direct link between exposure and adverse effects [Reed 1996c]. However, the evidence from epidemiological studies is limited because such studies cannot be controlled experimentally, and because it is based strictly on observation [Jardine 2004].

2.3.2 Dose-Response Assessment

Once information regarding the inherent toxicity of an agent is gathered, an analysis of the relationship between the type of dose received and the respective response is conducted. There are three different types of doses: external, internal and biologically effective doses.

External doses are the most far removed from driving the dose-response relationship and are also the most frequently estimated because they only require exposure concentrations and intake rates, which are generally available from toxicology studies. Internal doses are calculated using the external dose and the bioavailability factor[3]. In theory, the biologically effective dose is the measure of greatest value for

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[3] "A pollutant or other chemical is in a physical or chemical form that permits it to be eaten, breathed, or otherwise absorbed by living things. This is known as the bioavailability of a substance. The bioavailability factor simply estimates the proportion of the substance in the environment that is available to be absorbed. For example, a bioavailability factor of 0.5 means that 50% of the substance in the environment is available to be absorbed. The bioavailability factor is unknown for many compounds. In

modelling dose-response relationships, but it is rarely available for risk assessment because it is difficult to measure and has not been obtained in most animal bioassays [Jardine 2004]. Figure 2.2 illustrates the types of doses in the dose-response relationship.

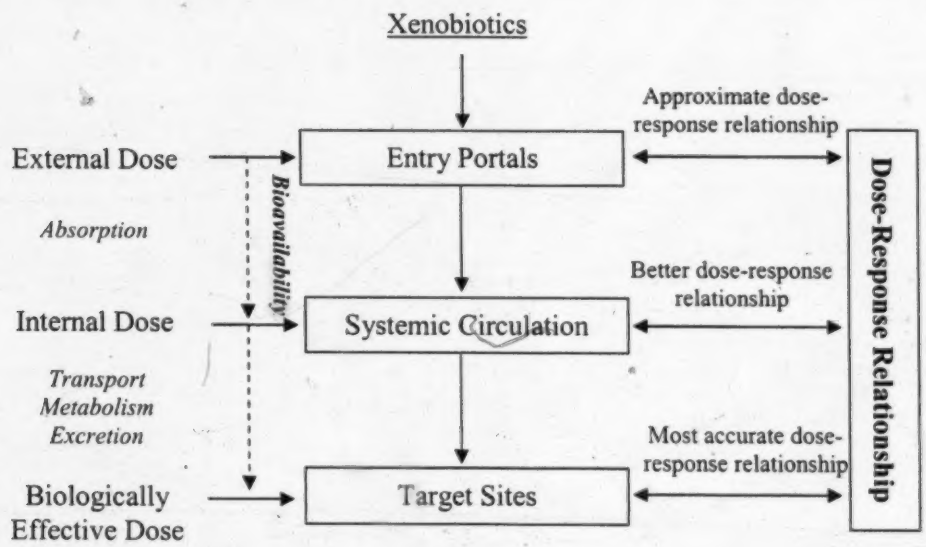

Figure 2.2 Illustration of the dose-response relationship under different types of doses[4]

Doses are usually normalized with respect to body mass as well as averaged over some exposure time, to get equation (3).

$$ED = \frac{C * CR * EF}{BW} \qquad (3)$$

where ED is the estimated dose and is generally measured in the number of milligrams of the contaminant that enter the body for each kilogram o body weight (mg/kg/day); CR is

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these cases it is assumed the bioavailability factor is 1.0 (i.e., 100% of the substance in the environment is available for absorption) [Health Canada 1995; p.58]."

[4] Source: Jardine 2004.

the contact rate, measured as the amount of water, food, air, etc., which is swallowed, inhaled or comes into contact with the skin in one day.; C is the exposure concentration; BW is the average body weight of an individual in kilograms; EF is an exposure factor, which indicates how often the individual is exposed during a year and the number of years that this pattern has been repeating itself. This factor is needed when exposure does not occur daily [Health Canada 1995].

The mathematical analysis of dose-response assessment takes the form of a dose-response curve. To determine if there is evidence of carcinogenicity, evidence in humans from epidemiological studies, evidence in short term *in vitro* tests, strength of evidence from animal toxicity studies, the type of tumor and frequency and the mechanism of action are evaluated. Dose-response curves are based on animal study data extrapolated to potential human responses using statistical principles or general theories of carcinogenesis. Dose-response curves for carcinogens are designed to pass through the origin on the basis that there is no threshold of response (i.e. exposure to one molecule of the carcinogenic substance can theoretically result in the DNA damage leading to a cancerous growth). The resultant curve displays carcinogen potency as the slope or grade of the curve [Reed 1996a]. The USEPA approach to the dose-response curve is to assume that there is no threshold whereby small doses elicit no response. That is, carcinogens have an effect at any dose. This is because cancer development is a multistage process that occurs over many years; therefore it could take multiple exposures to a number of agents before a cancerous growth occurs [Reed 1996a].

The multistage model for cancer recognizes that there are multiples stages of

development, so that the equation would look like (4):

$$P = 1 - \exp [-(q_0 + q_1 d + \ldots + q_k d^k)] \quad (4)$$

where d is dose and q terms are the empirical curve fit constants. This model is an exponential expansion which is fitted to limited data from carcinogen bioassays. To fit this model to a linearized multistage model, at low doses, the "d" terms raised to higher powers become insignificant, which gives equation (5).

$$P(d) = 1 - \exp [-(q_0 + q_1 d)] \quad (5)$$

If background risk is taken as P(0), then looking at excess risk [ER(d)] above background risk, equation (5) is converted to equation (6):

$$ER(d) = q_1 * d \quad (6)$$

where ER is the extra risk over background risk at dose d, giving the lifetime probability of cancer; d is the average daily dose of a carcinogen over a lifetime; and q_1* is the slope factor derived from the upper 95% confidence interval of the linearized multistage curve [Jardine 2004].

2.3.3 Exposure Assessment

Exposure assessment is used to evaluate the actual levels of exposure and absorption of the toxicant among a population of exposed individuals. Levels of exposure are based on frequency and duration of exposure as well as the levels of toxicant in the soil, water, air, and/or food. There are three routes of exposure: dermal, oral and inhalation. The concentrations of toxicant in the air must be related to how much an adult would absorb into their system in one day. This is done using the adult breathing rate of $20 m^3$/day.

Similar rates are available for water intake. The exposure duration is assumed to be 70 years and the frequency of residential exposure is 350 days per year [Reed 1996b].

2.3.4 Risk Characterization

Risk characterization requires assessing information obtained from the hazard identification, dose-response assessment and exposure assessment. Risk characterization allows risk values for carcinogens to be calculated by multiplying the lifetime average daily dose by the slope factor of the dose-response curve [Reed 1996d]. This thesis study uses risk characterization to quantify the risks to the farmer population of PEI due to concentrations of residues remaining in the air after spraying chemicals.

2.4 A DIGRESSION ON THE POTENTIAL BENEFITS OF GM TECHNOLOGY TO DEVELOPING COUNTRIES

This study focuses on the impacts of GM technologies on farmers in a developed country. However, there is considerable debate about the impacts on farmers in developing countries where the health benefits may be much larger. In this section the literature on benefits of GM technology to farmers in developing countries is reviewed.

2.4.1 Use of pesticides in developing countries: Yield Implications

There are a number of factors involved in the ability of farmers in developing countries to adopt safe pesticide technology. A number of studies have pointed out the constraints of lack of trained labour, power, and machinery; lack of knowledge; poor financial position of farmers; illiteracy; and a high cost of pesticides [Muhammad et. al. 1998, Samy 1998, Sivanarayana 2000]. The need for training of labour, specialized

equipment, and protective clothing to provide safer application and the high costs of pesticides are common barriers to the safe use of pesticides in these circumstances.

Lack of knowledge encompasses the small farmers' lack of access to or understanding of technological recommendations of the extension services and the lack of education and awareness of associated agricultural aids to growing their crops. Recommendations proposed by Muhammad et. al. (1998) are that private pesticide firms should put more effort into their demonstration plots, field days and publications; and that the extension service should organize short training, workshops and seminars for farmers in order to create awareness.

It could be inferred that by employing the use of pesticides, additional machinery and related specialized inputs, farmers in developing countries could have better control of pests and diseases in their crops. Training could enable farmers to do more scouting for pests and the onset of diseases as well as to combat these pests and diseases through the use of pesticides. With appropriate small-scale technology it could be possible to apply pesticides over wider areas, and improve yields. However, pesticides are an expensive input and often underused in developing countries, compared to many developed countries. Thus yield improvements as well as the possibility of reduced input costs may be achieved in these circumstances. Recent studies are demonstrating that this is the case with Bt cotton, which is now widely grown in many developing countries, and particularly in China.

For example in China, a developing country where Bt cotton has been grown for the past six years, it has been found by Pray et. al. (2002) that yields per hectare have

increased[5], pesticide costs have been reduced, as has the time spent spraying pesticides and the number of incidences of pesticide poisonings. The actual amount of pesticide use has been reduced by approximately 62% in growing Bt cotton versus growing non-Bt cotton [Mackey 2003]. Qaim and Zilberman (2003) have come to similar conclusions about growing Bt cotton in India. They conclude that on-farm field trials with Bt cotton on 395 farms in seven states show that pest damage is reduced, yields are increased and insecticide use on Bt cotton plots was reduced by almost 70%.

The new genetic technology has proven to be valuable to more than 4 million smallholders[6] in China, and although the increased yield has pushed prices down, prices are still sufficiently high for adopters of Bt cotton to make substantial gains in their net income [Pray et. al. 2002]. Pray et. al. hypothesize that China is similar to other developing countries, in particular India, where farmers face the same pests and pest resistance problems, suggesting that farmers in India could also make net gains from growing locally adapted Bt cotton varieties. It could also be inferred that farmers could make income gains with other genetically modified crops.

2.4.2 Use of pesticides in developing countries: Health Implications

Tests of pesticides carried out in 21 developing countries in Latin America, Africa and Asia from 1989 to 1994 showed that 34% of the tested product concentrations of active ingredients were outside the FAO tolerance limits [Kern & Vaagt 1996]. The

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[5] Yields have increased 10% in China over non-Bt cotton and 60% in Africa over non-Bt cotton [Mackey 2003].

pesticides contained active ingredients that were too strong and/or contaminated when compared to FAO specifications. Possible reasons for this occurrence were thought to be poor production and formulation methods, as well as poor labelling and packaging.

When comparing the pesticide formulations available in developed and developing countries, there was no evidence found that the formulations available in developed countries were outside the FAO tolerance limits and specifications. In the developing world, however, studies such as those done by Kern & Vaagt (1996) and Balasubramaniam (1997) show that the quality of available pesticides has not improved at the same rate.

Problems arise due to a lack of knowledge on the part of farmers in the developing world regarding pesticide application and storage. This is discussed further in the balance of this section, based on studies in developing countries on the use of pesticides and possible problems and solutions arising from their use.

Balasubramaniam (1997) reports that in Indonesia, Malaysia, the Philippines and Thailand certain pesticides are not registered because of their toxic properties, while others may be placed under severe handling and application restrictions. The results of these problems are seen in records from hospitals and surveys indicating that pesticide poisoning is a serious problem. The Food and Agriculture Organization (FAO) of the United Nations has issued circulars advising small farmers against using pesticides belonging to class Ia (extremely hazardous, including hexachlorobenzene) or Ib (highly

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[6] Smallholder generally refers to farmers who have 5 or fewer hectares under

hazardous, including numerous organophosphates and carbamates). These classifications are based on the World Health Organization hazard classifications[7]. A prior informed consent (PIC) procedure has also been implemented by the FAO and the United Nations Environment Programme. Another important area that the farmers need to be educated in is the level of pesticide residues, particularly in vegetables. Balasubramaniam (1997) asserts that many poor countries, including Indonesia, Malaysia, the Philippines and Thailand, do not have the necessary resources to carry out the testing and analyses of an adequate number of samples of pesticide formulations and their resulting residues. It is suggested that regional or subregional pesticide analytical facilities are needed.

In a study done in a rural agricultural community in Kenya [Kimani & Mwanthi 1995], the extent of use of agrochemicals by a sample of farmers was evaluated, for the years from 1987 to 1990. It was found that more than 95% of all farmers sampled used pesticides extensively. The risk of exposure to pesticides was found to be higher among women than men, and babies and children were concluded to be more at risk to exposure than were women. Additionally, knowledge and awareness in regards to handling and storage of agrochemicals was limited to some extent, leading to adverse health effects, ranging from acute to chronic conditions. Suicide attempts by ingestion of agrochemicals was prevalent [Kimani & Mwanthi 1995].

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development.

[7] WHO Recommended Classification of Pesticides by Hazard. Available Online: http://www.inchem.org/documents/pds/pdsother/class.pdf

From a survey conducted amongst sugarcane farmers in Fiji, Szmedra (1999) reported that farmers who used pesticides were found to have an increased incidence of chronic illness and disease when compared with a non-user control group. Even though average production of sugarcane was higher among pesticide users, the average annual medical costs were also significantly higher, as were the number of days missed due to illness.

The studies noted above all indicate a need for farmers, particularly in developing countries, to be better educated about the risks and benefits of using pesticides in their operations. This could be approached through a system of institutional support funded by the governments of those countries to provide advice on proper handling and application as well as providing information describing the health implications of pesticide exposure [Szmedra 1999], or by the FAO issuing information circulars as mentioned by Balasubramaniam (1997).

2.5 CONSUMER PERCEPTIONS OF GM TECHNOLOGY

One possible reason for slow consumer acceptance of GM food products may be the uncertainty or perceived risk of consuming genetically modified products. Numerous polls have assessed consumers' perceptions about food biotechnology. However, Hallman and Aquino (2003) suggest that since these were conducted by different researchers at different times, asking different questions and having different methods and sampling procedures, these may not be a good guide to accurate conclusions

about how public opinion changes over time, how opinions differ around the world or the basis for consumer's opinions.

Results from a survey completed by Hallman and Aquino (2003) show that US citizens have little awareness of GM food products, with only 12% saying they knew a great deal about GM. The authors indicate that this is consistent with other current surveys on consumer awareness of biotechnology. Furthermore, 62% of respondents say they never have conversations about genetic modification and 55% of respondents know "very little" about GM technology.

Hallman and Aquino translate the apparent lack of awareness and knowledge regarding biotechnology and GM food into a general lack of recognition that GM food products are on the supermarket shelves (only 52% of respondents were aware that GM food products are currently being sold in United States supermarkets). Support for genetic modification of plants was higher than that for animals, which is consistent with other surveys. However, public support for plant and animal-based GM foods had declined between 2003 and 2001, as had the conviction that GM technology would improve the quality of respondents' lives [Hallman & Aquino 2003]. These conclusions are consistent with those drawn from other polling studies.

From a survey by Hossain et. al. (2002), examining the relationship of respondents' socio-economic characteristics and personal values on their willingness to purchase GM foods, the conclusion is derived that consumer acceptance critically depends on education, knowledge, trust and confidence in regulatory agencies and religious views, among other characteristics. That study analyzes willingness to purchase

GM products that do not confer any additional benefits in relation to their non-GM counterparts.

2.6 SUMMARY

This chapter has explained the cost-benefit analysis technique, the cost of illness technique, the value of a statistical life approach and how these techniques can be combined to estimate the monetary values of nonfatal and fatal cancers. Health risk assessment procedures were outlined in regards to hazard identification, dose-response assessment, exposure assessment and risk characterization. A review of literature outlining the potential benefits of GM technology to developing countries has been presented, as have consumer perceptions about GM technology. In the next chapter, the genetic modification of potatoes, potato cultivation methods and the pesticides used in PEI potato production will be addressed.

CHAPTER 3: THE POTATO: GENETIC MODIFICATION, CULTIVATION PROBLEMS AND PESTICIDES

3.1 GENETIC MODIFICATION OF POTATOES

Monsanto had produced three types of genetically modified potatoes by 1999. This section concentrates on the two approved for and actually grown in Canada. The NewLeaf Russet Burbank potato was approved for growth in 1996, after which potato farmers started growing this potato variety. The NewLeaf potato contained two genes that differed from those of conventional potatoes. These genes produced two proteins, the first derived from the bacteria *Bacillus thuringiensis* (Bt), which protected against the Colorado potato beetle, while the second was a biological marker that allowed researchers to identify the modified potatoes.

With the introduction of resistance to the Colorado potato beetle in the NewLeaf potato, adopting farmers found that they sprayed fewer chemicals and that the plants were healthy and bushy throughout the growing season. However, it was reported that when farmers went to harvest the potatoes from the first GM potato crop, they were disappointed with the number of tubers under each hill. Those farmers that planted the potatoes again the next year made slight adaptations in how they planted and grew the modified potatoes, such as plant spacing, distance between the rows, and the depth to which the seed was planted. These changes were made on the recommendation of the PEI potato specialist and the Cavendish Farms Crop specialist, who grew test plots of the same variety. A higher yield was noticed, while the aesthetic benefit of healthy looking,

bushy plants was maintained. These small adaptations to each of the different climates in PEI meant that the producers who continued to grow the potato after the disappointing initial growing year realized similar yields and tuber quality and a reduced amount of pesticides sprayed by the end of the 1999 growing season [MacPhail 2003, Coffin 2003].

The NewLeaf Plus Russet Burbank potato was being grown in the United States and was approved for use in Canada in October, 1999. This potato was modified to be resistant to Colorado potato beetle as well as to the infection of the potato leafroll virus. The only change in the NewLeaf Plus potatoes was the addition of viral sequences of potato leafroll virus, which "exhibit the trait of resistance to infection and subsequent disease caused by potato leafroll virus through an incompletely understood process which may involve silencing of viral gene translation, which has been termed 'replicase-mediated resistance' [Health Canada 1999a]." The production of the potato leafroll virus protein was undetectable in either leaf or tuber tissue and was therefore considered to be at a lower concentration than for plants naturally infected with potato leafroll virus [Health Canada 1999a].

The NewLeaf Pro Russet Burbank potato that was being developed and was scheduled for release in 2002 was modified for resistance to Colorado potato beetle, potato leafroll virus, potato virus Y, and late blight. The resistances to Colorado potato beetle and potato leafroll virus were being developed in the same way used for the NewLeaf Plus potato. Potato virus Y resistance was to be developed by including the coat protein from the ordinary strain of potato virus Y. (Potato virus Y is an aphid transmissible RNA virus that commonly infects potato crops, causing serious disease and

economic loss.) The potato virus Y resistant trait exhibited resistance to infection and subsequent disease through a process related to viral cross-protection [Health Canada 1999b]. It is unclear whether Monsanto had discovered how to develop the late blight resistance when its potato research programs stopped in 2001.

3.2 POTATO PESTS, DISEASES AND VIRUSES[8]

3.2.1 Insect Pests

Insect pests reduce yields and spread virus diseases by feeding on potato plants and tubers. It has been estimated that without pesticide controls, approximately 30% of the crop would be lost each year and that with controls, less than 3% of the crop is lost to insects [Advisory Committee on Potatoes 1993]. The major insect pests affecting PEI potato production are Colorado Potato Beetles (CPB), European Corn Borer and aphids.

Colorado Potato Beetle

The Colorado Potato Beetle reached PEI by 1883. Its hosts are limited to plants belonging to the family Solanaceae, preferring potatoes, but also attacking eggplant, tomato, and weeds like goundcherry, wild tomato and climbing nightshade. The CPB cannot be confused with any other beetle in Canada.

The adult beetle over-winters in the soil. As temperatures increase in the spring, the beetles move upward in the soil and first appear in the last week of May or early June, immediately seeking host plants. They feed for a few days, mate and then lay 300-500

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[8] This section was adapted from Advisory Committee on Potatoes (1993).

eggs between June and late July. Due to the development of the beetles and the nature of the weather in PEI, there is normally only one generation of the beetle in any given year.

The beetles cause crop injury by feeding on the foliage and plant stems. If there are high populations of this insect in a field, it can completely defoliate the plants. Extensive beetle feeding at any time through the season, especially when the crop is in bloom can reduce yield because of the reduction in leaf surface decreasing the ability of potato plants to produce nutrients for storage in the tubers.

European Corn Borer

The European corn borer is known to feed on over 200 different species of plants including corn, potatoes, beans, beets, and peppers. Growers in PEI started to complain about its presence in the early 1990s [Advisory Committee on Potatoes 1993]. The European corn borer over-winters as fully grown larvae in the stem of its host plant. The larvae emerge as adults around the middle of July on PEI. Eggs are laid on the undersides of leaves and hatch in three to nine days. The hatched larvae feed on the leaves for a few days, then bore into the stem of the plant, completing their development and preparing for over-wintering.

The larva damages the crop by entering the stem and destroying the pith, xylem and phloem. These damaged plants are susceptible to wind damage, water stress and invasion from pathogens. Impacts on the potato crop appear to be a function of larval numbers, the developmental stage the plant is in at the time of attack, the cultivar, presence of other insect pests and diseases, and other stress factor such as temperature, water and fertility. The window of opportunity for destroying larvae is limited, as they

are only on the outside of the plant for a few days. Insecticides are not effective after the larva enters the stem [Advisory Committee on Potatoes 1993].

Aphids

Three different types of aphids are a problem in PEI. These are the green peach aphid, the buckthorn aphid, and the potato aphid. The green peach aphid attacks potatoes late in the season and its populations rarely increase sufficiently to weaken the crop and reduce yields. It over-winters in peaches, Canada plum and black cherry, and its summer hosts include many plant families. Among vegetable crops, the potato is a preferred summer host [Advisory Committee on Potatoes 1993].

The green peach aphid gives birth to live young without mating during the growing season. As the season ends, sexual forms appear, mate and lay eggs. The aphids produced in this manner are the ones to over-winter in hosts. The green peach aphids cause crop injury by feeding on the lower leaves, flowers and shoot tips of the potato. This type of aphid is the most important vector of potato leaf roll virus (PLRV) and it is an effective vector of potato virus A, potato virus S, and potato virus Y (PVY) [Advisory Committee on Potatoes 1993].

The buckthorn aphid over-winters in one of several species of buckthorn, with secondary hosts include some species of the Solanaceae family, among others. It is the smallest of the three aphids. The winged males and females reproduce in late summer, after colonizing weeds and potato crops and migrating back to buckthorn. The winged females give birth to wingless sexual forms which in turn mate and lay the over-wintering eggs [Advisory Committee on Potatoes 1993].

The buckthorn aphid infests the lower leaves of potatoes, but rarely increases in numbers sufficiently to weaken a crop or reduce potato yields. In a dry year, it can spread throughout a field and is an effective vector of PVY.

The potato aphid is normally the first aphid to appear on potatoes in PEI, showing a preference for the middle and upper part of the plant. High numbers can weaken the crop and reduce yields. The potato aphid causes injury to potato crops by attacking the flowers and shoots. The presence of the potato aphid can be seen by the wilting of the plants and honeydew on the leaves. It rarely transmits PVY to potatoes, though it can to tobacco, and is a poor vector for potato virus A and PLRV [Advisory Committee on Potatoes 1993].

3.2.2 Diseases

The two different types of disease that can affect a potato crop are fungal and bacterial diseases. The bacterial diseases can cause much crop damage, however, the fungal diseases are normally more devastating. The main fungal diseases of concern in PEI are late blight and early blight [Advisory Committee on Potatoes 1993].

Late Blight

Late blight is caused by a fungus and is a destructive disease in the Atlantic region of Canada. Disease symptoms usually appear on older (lowest) leaves soon after flowering, following wet or overcast weather conditions. It appears as dark green, water soaked areas at the leaf margins that spread inwards and later become dark brown and brittle. The edge of this has a fluffy white growth (mildew) visible on the underside of infected leaves when humidity is high or in the morning following a dew. The mildew

produces sporangia which are spread by wind or rain to other plants and when conditions are wet or humid, the spread of disease can be extremely rapid, causing progressive defoliation, yield loss, tuber infections and plant death. Storage of infected tubers can cause the fungus to be spread to health tubers, resulting in further crop loss [Advisory Committee on Potatoes 1993].

The fungus can only over-winter in living potato tissue. It is therefore important that infected tubes not be used for seed or discarded into cull piles, or that "volunteer" potatoes not be left in the field to over-winter and produce the fungus in the next growing season. In order to control the spread of the disease, cull piles must be destroyed by composting and volunteers in the field must be buried or frozen with herbicides and proper crop rotation practices. The most important control is to use disease-free seed [Advisory Committee on Potatoes 1993].

Foliar fungicides, such as Bravo, Dithane or Manzate, provide effective disease control when applied at the proper rates and when used with disease forecast announcements and crop inspection reports. It is noted in the publication *Atlantic Canada Potato Guide* that some fungicides are systemic and curative, but that the continued use of these fungicides can lead to resistance to the fungicide in the pathogen.

Early blight

The fungus that causes early blight occurs wherever potatoes are grown. Crop losses occur if heavy foliar infection takes place around the time of flowering. The disease appears as small, brown pinhead dots on older leaves, which enlarge as

concentric rings, causing extensive loss of leaf area and consequent yield reductions. Disease development is prominent when alternating wet and dry periods are observed. The fungus survives in crop residue, soil, infested tubers and on other hosts [Advisory Committee on Potatoes 1993].

The use of high quality, disease-free seed and not using potatoes for at least two years (preferable three) in crop rotations are the advised ways to ensure that successive outbreaks do not occur. Foliar fungicides are available for control and should be applied as soon as the disease appears. Several applications may be necessary when weather conditions are favourable for disease development [Advisory Committee on Potatoes 1993].

3.2.3 Viruses

The two main viruses in PEI potato production are potato virus Y and potato leaf roll virus. Viruses rarely affect the eating quality of the tuber, though they remain in the tuber themselves and are consumed, as well as resulting in yield loss. The viruses transmitted through seed tubers can spread extensively from plant to plant in the field by aphids, which feed by puncturing the leaf or stem and sucking sap [Advisory Committee on Potatoes 1993].

Because aphid populations rarely manage to over-winter in Atlantic Canada, the time when they do appear is later in the season. Potato plants tend to become more resistant to the effects that aphids have on them as they mature. It is due to this that PEI has a high reputation as a seed-growing region. However, when aphids develop in larger

numbers or come earlier in the season, there can be major problems [Advisory Committee on Potatoes 1993].

Mosiac Viruses (PVY)

PVY causes mosiac-type symptoms on susceptible potato varieties, which is accompanied by a dropping of the lower leaves. The mosiac viruses are non-persistent in that the aphids which carry and transmit the virus (on their stylets) can only retain it for a short period of time. The virus is spread largely by winged aphids, very few of which bring the virus with them, but pick this up from diseased plants and pass it on to healthy ones as they move from plant to plant, probing with their stylets for a place to feed [Advisory Committee on Potatoes 1993].

Potato Leaf Roll Virus (PLRV)

PLRV causes loss of chlorophyll, rolling of the leaves and often severe stunting of the plant. Yield can be significantly reduced by this virus. It is transmitted by aphids, which ingest the virus into their stomach and later regurgitate it in saliva after moving to feed on a health plant. Aphids remain infected for their entire lifespan [Advisory Committee on Potatoes 1993].

The two main sources of infection of potato leafroll virus are diseased plants in other fields, quite possibly hundreds of miles away, because aphids can carry PLRV long distances if wind conditions are favourable and there are diseased plants within the field. Aphids that do not have the virus when they arrive can pick it up from a diseased plant or they can produce young on diseased plants, that acquire the disease as they develop

[Advisory Committee on Potatoes 1993].

3.3 PESTICIDES: CHEMICAL FAMILIES AND THEIR RISKS

In Canada, pesticides have to be registered under the federal Pest Control Products Act (PCPA). The current PCPA was passed in 1969 and has not been significantly amended since. There are approximately 6000 pesticide products registered for use in Canada, translating into 50 million kilograms of herbicides, insecticides and fungicides applied each year at a cost of one billion dollars. These products are used in agriculture, for lawn care, in fish farming, in homes, on golf courses and in industry [WWF date unknown].

There are 335 pesticide products registered for use in PEI, approximately 100 of which are used in potato production. These products consist of 28 active ingredients commonly found in insecticides and 11 active ingredients commonly found in fungicides. The active ingredients fall into the following main classes of chemicals: organophosphates, pyrethroids, ethylene bisdithiocarbamates (EBDCs), chloro-nicotinyl, carbamates, organochlorines and botanical insecticides [Reeves 2002].

3.3.1 Organophosphates:

All organophosphates are considered neurotoxic because they may cause irreversible cholinesterase inhibition, which means they increase the number of nerve impulses, leading to an over-stimulation of the nerves and eventually death. They were used in the Second World War as neurotoxins. There are a number of insecticides with active ingredients belonging to the organophosphate family, including diazinon,

methamidaphos, dimethoate, malathion, methidathion, azinphos-methyl, and chlorpyrifos [HIARC 1998].

Besides causing cholinesterase inhibition, organophosphates may also cause neuropathology of the visual system or effects on cognitive functions [HIARC 1998]. Effects on animal fetuses were seen following pre and/or post natal exposure and most often manifested as decreased pup viability at doses that caused considerable cholinesterase inhibition in the parental animals [HIARC 1998]. These results were regarded as the susceptibility of young animals to their environment and not seen as developmental toxicity. For most organophosphates, "fetal effects were seen at the same dose that cause maternal toxicity" [HIARC 1998; p.5].

Acute effects of poisoning occur within hours of exposure and, for mild to moderately severe poisoning, include tightness in the chest, wheezing, headache, insomnia, slurred speech, tremor, muscle weakness with cramping, increased sweating, salivation, nausea, vomiting, cramps, watery diarrhea and involuntary defecation/urination. Pupils may become constricted. Death due to organophosphate poisoning typically results from cardiac or respiratory failure [Dyro 2001].

In 1983, the US mortality rate from non-intentional pesticide poisoning was 2.7 per 10 million men and 0.5 per 10 million women. On average, approximately 20,000 cases of organophosphate poisonings are reported each year, with 2.3-2.6 per 10 million of the cases representing suicidal ingestion [Dyro 2001]. According to the American Association of Poison Control Centers, there were 16,392 reported exposures to organophosphates, with 11 reported deaths in 1998. This is likely to be an underestimate

of organophosphate poisonings as many cases of cumulative exposure in agricultural workers go unreported [Dyro 2001].

In addition to their roles as active ingredients in pesticides, organophosphates are currently being used as therapeutic drugs for a variety of dementia diseases, including Alzheimer's, schistosomiasis and primary degenerative dementia [Dyro 2001].

3.3.2 Pyrethroids

Pyrethroids disrupt the endocrine system by mimicking estrogen, causing excessive levels of estrogen in females and males. Pyrethroids are known carcinogens, and the extra estrogen in humans can lead to cancerous breast tissue in both males and females. There is evidence that pyrethroids can harm the thyroid gland. Pyrethroid derivatives can have neurotoxic effects, especially on children [Kaplan, 2000]. Insecticide active ingredients that belong to this class are deltamethrin, permethrin, and cypermethrin [EXTOXNET 1993a, EXTOXNET 1993b, EXTOXNET 1995].

Inhaling high levels of pyrethroids may result in asthmatic breathing, sneezing, nasal stuffiness, headache, nausea, incoordination, tremors, convulsions, facial flushing and swelling, and burning and itching sensations. Recovery from serious poisoning in mammals is fairly rapid. Dermal absorption in rats and rabbits requires large doses, however, on broken skin, pyrethroids produce irritation and sensitization, which is further aggravated by sun exposure [EXTOXNET 1994].

Pyrethroids are semisynthetic derivatives of pyrethrins, which are natural insecticides produced by certain species of the chrysanthemum plant. Pyrethrins are made by harvested the flowers of the plant and are either dried and powdered or the oils

are extracted from the flowers. The yield of the resulting active ingredient content is usually about 30%. "Pyrethrin dusts" are used to control insects in horticultural crops and since they are only 0.3-0.5% pyrethrins, are applied at rates of up to 50 lb/acre [EXTOXNET 1994].

The natural pyrethrins are contact poisons that quickly penetrate the nerve system of the insect. A few minutes after application, the insect cannot move or fly away. However, enzymes in the insect swiftly detoxify the natural pyrethrins. Thus, some pests will recover. To delay the enzyme action so a lethal dose is assured, organophosphates, carbamates, or synergists may be added to the pyrethrins. The pyrethroids tend to be more effective than natural pyrethrins in killing insects while they are less toxic to mammals [EXTOXNET 1994].

3.3.3 Ethylene bisdithiocarbamates (EBDCs)

The EBDCs are considered to have low short-term mammalian toxicity; however, a major concern with EBDCs is the breakdown of the chemical into ethylenethiourea (ETU) [EXTOXNET 1996c]. ETU can cause thyroid problems in laboratory animals and is considered by the United States EPA to be a *probable* but not proven human carcinogen [EXTOXNET 1996c]. Fungicide active ingredients from this class are mancozeb and zineb [Reigart & Roberts 1999].

Occupational exposure can occur through inhalation and dermal contact. The general population may be exposed to ETU via inhalation of ambient air, ingestion of drinking water, and dermal contact with this compounds containing ETU. Pesticide food monitoring data show that the general population may be exposed to ETU by the

ingestion of some food products. There is no range of toxicity that can be established for this family of chemicals at this point in time [CASRN 1996].

3.3.4 Chloro-nicotinyl

Chloro-nicotinyl interferes with the transmission of stimuli in the nervous system, by causing a blockage in the nicotinergic pathway of the brain, which is more abundant in insects than it is in mammals [EXTOXNET 1996b]. This leads to an accumulation of acetylcholine (cholinesterase inhibition), in much the same way as for organophosphates. Imidacloprid, a member of this chemical family, is a commonly used active ingredient in PEI potato production for combating aphids, thrips and some beetles, and is considered to be one of the insecticides used in largest volume [Cox 2001].

3.3.5 Carbamates

Carbamates work in much the same way as organophosphates by causing an inhibition of cholinesterase, however, this inhibition is reversible. The active ingredients of insecticide included in this class are methomyl, oxamyl, carbofuran and carbaryl [NJDEP 1999]. Short term health hazards of carbamates include absorption through the skin, especially through cuts and scratches and eye irritation. Known long term health hazards include carbaryl and carbofuran plus elements in the human stomach which can combine to form possible cancerous chemicals; carbofuran has been shown to cause birth defects or reproductive problems in laboratory animals [EXTOXNET 1996a].

Carbamates have a wide variety of symptoms of poisoning, including headache, dizziness, extreme weakness, twitching or tremor, slow heartbeat, swelling or tightening in the chest, sweating and nausea. Effects to the eyes can include blurred or dark vision,

redness, extensive tearing, discomfort in the eye muscles, and changes in the size of the pupils [NJDEP 1999].

3.3.6 Organochlorines

Short-term effects from organochlorines affect the nervous system, where the compounds induce a hyper-excitable state in the brain [Cox 1997]. Organochlorines may also affect the endocrine system. DDT and PCBs (polychlorinated biphenyls) are considered to be a part of this chemical class. The insecticide active ingredient methoxychlor and the fungicide active ingredients chlorothalonil and endosulfan are in this class.

Persons exposed to high levels of organochlorine pesticides by any route should be observed for sensory disturbances, incoordination, speech slurring, mental aberrations, and involuntary motor activity that would warn of imminent convulsions [Reigart & Roberts 1999].

3.4 SUMMARY

It has been illustrated that the pests, viruses and diseases that are commonplace in PEI potato production are a possible impediment to growing a profitable crop in successive years. The chemical families that each pesticide active ingredient belongs to have been discussed in relation to the potential health risks that could occur when they are used to control these pests, viruses and diseases. In the next chapter, a spraying schedule using

the more common active ingredients is presented as are the financial costs of using these chemicals.

CHAPTER 4: PARTIAL BUDGET ANALYSIS: DATA AND RESULTS

4.1 ACTIVE INGREDIENTS IN PEI POTATO PESTICIDES

There are 28 active ingredients commonly found in insecticides and 11 active ingredients commonly found in fungicides that are approved for use in PEI potato production.

In interviews with farmers and the PEI provincial potato specialist (conducted in Summer 2003), it was determined that the most commonly used pesticides are Senator, Admire, Lorox, Ripcord, Monitor, Dithane, Bravo, and Reglone. The interviews were conducted amongst a sample size of seven, consisting of five farmers, one crop specialist at Cavendish Farms and the provincial PEI Potato Specialist.

Respondents were first asked general questions about the type of farm they operated, how long they had been involved in operating the farm, and if they had implemented processes that reduced pesticide applications, storage costs or increased yields, and if so, were they able to maintain the processes. They were then asked about their spraying schedules, application rates, and cost of application for conventional potatoes, and then were asked the same questions as they related to NewLeaf potatoes. Respondents were also asked if they perceived any benefits from growing NewLeaf potatoes, to which their responses were aesthetic benefits and time/cost savings benefits[9]. Table 4.1 summarizes information provided by the respondents and shows the pesticides

that would be used in a typical growing season and their uses, as well as the recommended amount to be sprayed in each application according to pesticide label instructions[10].

Table 4.1: Pesticides used in a typical potato growing season, their uses, and manufacturers' recommended spraying amounts

Pesticide	Active Ingredient	Controls	Application Rates per hectare
Senator	Thiophanate-Methyl	seed piece decay	0.5 kg/100 kg seed
Admire	Imidacloprid	aphids & beetles	0.85 L/ha
Lorox	Linuron	annual broad leaf weeds	2.2 kg/ha
Ripcord	Cypermethrin	corn borer	0.0625 L/ha
Monitor	Methamidophos	Colorado potato beetle, aphids	1.75 L/ha
Dithane	Mancozeb	early/late blight	1.1 kg/ha
Bravo	Chlorothalonil	early/late blight	1.2 L/ha
Reglone	Diquat	Topkiller	1.25 L/ha

[9] Farmers found that they sprayed less often for CPB, which meant a cost savings from reduced pesticide use and a time savings of at least four hours per spray, depending on farm size.

[10] Note that two versions of the partial budget of pesticide applications and related costs for various potato varieties were calculated - one with the spraying recommendations set out by the pesticide labels and the other with the recommendations of the provincial potato specialist. The two differed almost exclusively in terms of pesticides that are not used in the typical spraying schedule. This thesis will deal only with the partial budget derived with spraying recommendations set out by pesticide labels.

4.2 SPRAYING RECOMMENDATIONS

The spraying schedule displayed in Table 4.2 was developed in consultation with the PEI Provincial Potato Specialist and encompasses the applications needed to combat the most popular pest, virus and disease problems encountered in a given year. The recommendations in this table are a "best guess" spraying schedule for NewLeaf Plus and NewLeaf Pro potatoes as the former were only grown for one year (in 1999) and the latter was not fully developed and therefore never cultivated. It should also be noted that the potato fields would not always be sprayed exactly in this manner in consecutive years, due to variations in weather and associated differences from year to year in pests, viruses and diseases. This table can be viewed as a "typical" annual spraying schedule, treating the fields for Colorado Potato Beetle, corn borer, and early and late blight, among other field problems.

Table 4.2: Frequency of Pesticide Sprays under Conventional and NewLeaf growing practices[a]

Type of Potato	Conventional	NewLeaf	NewLeaf Plus	NewLeaf Pro
Chemicals Sprayed	Senator	Senator	Senator	Senator
	Admire (IF)[b]	Lorox	Lorox	Lorox
	Lorox	Ripcord	Ripcord	Ripcord
	Ripcord	Monitor	Bravo	Dithane
	Monitor	Bravo	Bravo	Dithane
	Bravo	Bravo	Bravo	Dithane
	Bravo	Bravo	Bravo	Reglone
	Bravo	Bravo	Dithane	Reglone
	Bravo	Dithane	Dithane	
	Dithane	Dithane	Dithane	
	Dithane	Dithane	Dithane	
	Dithane	Dithane	Dithane	
	Dithane	Dithane	Dithane	
	Dithane	Dithane	Dithane	
	Dithane	Dithane	Dithane	
	Dithane	Dithane	Reglone	
	Dithane	Reglone	Reglone	
	Reglone	Reglone		
	Reglone			

[a] Each occurrence of the pesticide in the spraying schedule indicates one spray for preventative control

[b] IF means that the pesticide is plant in furrow with the potato seed. Since the NewLeaf brand had Colorado Potato Beetle resistance built into their potato seed, this application was no longer necessary

4.3 PARTIAL BUDGET DEVELOPMENT

The costs associated with the purchase of containers of pesticide were obtained from Cavendish Agri-Services, a privately owned Atlantic Canadian business specializing in the sale of fertilizer and crop protectants to the agricultural sector in Atlantic Canada and Maine in September 2003. Dividing the cost of the container of the

specified pesticide products by the size of the container and multiplying by the recommended amount to be sprayed formed the basis of calculation of this component of the cost per spray.

$$\text{Ingredient Cost/Spray/ha} = \frac{\$ \text{ of Container}}{\text{Volume of Container}} * \text{Recommended Application} \quad (7)$$

This figure was adjusted to include application costs, in the form of the rental cost of the machinery needed to spray the chemicals. The rental cost of machinery reflects the depreciation of the equipment, gasoline costs, and an estimate of the cost of time required for the farmer to operate the machinery.

It was assumed that 40 acres could be sprayed in one hour. The tractor rental rate was taken as $28 per hour and the pesticide sprayer rental rate as $15 per acre [Kensington Agriculture 2004]. The cost per acre of spraying per se was calculated according to equation (8):

$$\text{Non-chemical costs per acre} = \frac{1 \text{ hour}}{40 \text{ acres}} * \frac{\$28.00}{1 \text{ hour}} + \$15.00 = \$15.70/\text{acre} \quad (8)$$

The non-chemical costs of chemical application per acre are converted to costs per hectare, using the conversion factor of 1 hectare equals 2.4710538 acres, giving the non-chemical application cost estimate per hectare of $38.80. This estimate is added to the chemical cost of spray per hectare to give the estimated total cost of spraying for each application of pesticide.

Table 4.3: Cost of spray per hectare of the pesticides listed in Table 4.2

Chemical	Cost of Spray
Senator	$100.30
Admire (IF)	$172.97
Lorox	$103.16
Ripcord	$46.04
Monitor	$88.71
Bravo	$51.55
Dithane	$46.45
Reglone	$66.68

The spray cost estimates in Tables 4.2 and 4.3, combined with the seed costs of the different potato varieties are the basis of the partial budget estimates, which provide the estimated change in farmers' costs per hectare that would occur if the NewLeaf potatoes were to be grown rather than conventional potatoes.

Table 4.4: Partial budget of farm input costs for potatoes based on manufacturers' spraying recommendations, PEI provincial potato specialist spraying schedule, and other application costs as described in section 4.3[a]

Conventional	Cost/ha	New Leaf	Cost/ha	New Leaf Plus	Cost/ha	New Leaf Pro	Cost/ha
Senator	$100.30	Senator	$100.30	Senator	$100.30	Senator	$100.30
Admire (IF)	$172.97	Lorox	$103.16	Lorox	$103.16	Lorox	$103.16
Lorox	$103.16	Ripcord	$46.04	Ripcord	$46.04	Ripcord	$46.04
Ripcord	$46.04	Monitor	$88.71	Bravo	$51.55	Dithane	$46.45
Monitor	$88.71	Bravo	$51.55	Bravo	$51.55	Dithane	$46.45
Bravo	$51.55	Bravo	$51.55	Bravo	$51.55	Dithane	$46.45
Bravo	$51.55	Bravo	$51.55	Bravo	$51.55	Reglone	$66.68
Bravo	$51.55	Bravo	$51.55	Dithane	$46.45	Reglone	$66.68
Bravo	$51.55	Dithane	$46.45	Dithane	$46.45		
Dithane	$46.45	Dithane	$46.45	Dithane	$46.45		
Dithane	$46.45	Dithane	$46.45	Dithane	$46.45		
Dithane	$46.45	Dithane	$46.45	Dithane	$46.45		
Dithane	$46.45	Dithane	$46.45	Dithane	$46.45		
Dithane	$46.45	Dithane	$46.45	Dithane	$46.45		
Dithane	$46.45	Dithane	$46.45	Dithane	$46.45		
Dithane	$46.45	Dithane	$46.45	Reglone	$66.68		
Dithane	$46.45	Reglone	$66.68	Reglone	$66.68		
Reglone	$66.68	Reglone	$66.68				
Reglone	$66.68						
Cost of Seed/ha[b]			$54.40		$54.40		$54.40
Total cost of pesticide application per hectare	**$1,222.30**		**$1,103.73**		**$1,015.02**		**$576.59**

[a] Non-chemical application cost estimates per hectare are $38.80, taking into account tractor rental and pesticide applicator rental.

[b] Seed costs reflect the royalty that Monsanto charged farmers in order to use NewLeaf seed, so the relative cost of conventional seed can be taken as zero.

4.4 IMPLICATIONS OF THE PARTIAL BUDGET

As the partial budget of Table 4.4 illustrates, the recommended pesticide applications for the genetically modified potatoes (based on pest controls engineered into these modified potatoes) vary only slightly between NewLeaf and NewLeaf Plus potato varieties. The recommended cultivation practices only reduce the number of pesticides applied by a minimum of one and two sprays respectively. Nonetheless, although the cost of purchasing modified seed increased farmers' costs by $54.40 per hectare, the decrease in the cost of applying chemicals results in a savings of $118.57 per hectare when growing NewLeaf potatoes and a savings of $207.28 per hectare when growing NewLeaf Plus potatoes, when compared to conventional potatoes. The aggregate cost implications are indicated in Table 4.5.

Table 4.5: Per hectare and overall savings of pesticide application costs compared to growing conventional potatoes

	NewLeaf	NewLeaf Plus	NewLeaf Pro
Savings per hectare	$118.57	$207.28	$645.71
Total savings[a]	$6,639,920.00	$11,607,680.00	$36,159,760.00

[a] Based on 1/10 of PEI's land base being devoted to growing potatoes in any year, or 56 000 hectares

As can be seen from Table 4.5, the potential to save between $6 million and $36 million on the cost of pesticide applications applies to the entire potato growing area of PEI, based on the assumption that genetically modified potatoes would be planted on all 56,000 hectares and that the spraying schedule given in Table 4.2 prevailed.

CHAPTER 5: ESTIMATION OF HEALTH RISKS: MODEL DEVELOPMENT

5.1 INFORMATION REQUIRED TO QUANTIFY HEALTH RISKS

In order to be able to quantify health risks from inhalation of chemical residues remaining in the air, chemicals that cause adverse health effects had to be identified. An additional requirement was for information on dose-response functions with which exposure estimates could be transformed into risk estimates. Each of the active ingredients in the pesticides listed in Table 4.2 was evaluated for this purpose.

The chemicals were evaluated using the detailed information on pesticides from two websites. Chemicals were initially assessed in the light of information on the Extension Toxicology Network website, a database service which is affiliated with Cornell University. This website provided the initial information needed to determine if there is danger to human health from the active ingredients, or from any of the inert substances added to the active ingredient in the pesticide formulations, or their metabolites. Information from this source is given in Table 5.1.

Table 5.1: List of identified dangerous active ingredients, their origins and summary of properties

Active ingredient	Pesticide	Pesticide Control	Dangerous chemical	Summary
Mancozeb	Dithane	Early/Late Blight	Ethylenethiourea	Metabolic by-product; EPA classified as probable human carcinogen
Chlorothalonil	Bravo	Early/Late Blight	Hexachlorobenzene	inert ingredient contaminating chlorothalonil formulations; EPA classified as probable human carcinogen[a]
Cypermethrin	Ripcord	Corn Borer	Cypermethrin	active ingredient; EPA classified as possible human carcinogen

[a] Source: Cox 1997

Once these chemicals were identified, the United States Environmental Protection Agency website was consulted to determine if research had been done on these chemicals that had led to an estimation of a dose-response function. The only identified dangerous chemical for which that information was available was hexachlorobenzene on the Integrated Risk Information System section of the USEPA website The USEPA has classified hexachlorobenzene as a probable human carcinogen on the basis that, when administered orally, it has been shown to induce tumors in the liver, thyroid and kidney in three rodent species. However, they have deemed that the reported epidemiological studies of hexachlorobenzene have not been designed to measure increases in cancer incidence as an endpoint and are inadequate in this context. The dose-response slope

(potency) factor has been calculated as 1.6 per (mg/kg)/day. Based on this slope factor, the environmental concentrations corresponding to various specified risk levels for inhalation exposure have been calculated as shown in Table 5.2.

Table 5.2: Dose-response function for inhalation exposure to hexachlorobenzene

Risk Level	Concentration
E-4 (1 in 10 000)	2E-1 ug/m^3
E-5 (1 in 100 000)	2E-2 ug/m^3
E-6 (1 in 1 000 000)	2E-3 ug/m^3

Source: IRIS 1998

Since no comparable documents could be found for cypermethrin and ethylenethiourea, although these are also considered to be dangerous chemicals, the dose response function for hexachlorobenzene was assumed to apply to these chemicals and used in the analysis which will be described in section 5.2. The actual dose-response functions for these chemicals could be more or less toxic.

The next step in the analysis to determine if there are health impacts from the use of these chemicals was to find a basis to estimate the levels of exposure of dangerous ingredients that an applicator could be exposed to. Estimates on which this is based is a study of pesticide residues remaining in the air after a single application of an identified pesticide, which is reported in Kollman (1995) – Summary of Assembly Bill 1807-3219 Pesticide Air Monitoring Results Conducted by the California Air Resources Board 1986 to 1995. The estimates from this study are reported in Table 5.3.

Table 5.3: Summary of Air Monitoring Results in micrograms per cubic metre (ug/m^3) after an application of chlorothalonil

Site	Sampling Interval[a]							
	1	2	3	4	5	6	7	8
1East-1	ND[b]	0.158	0.122	0.098	0.026	0.043	0.051	0.034
1East-2	ND	0.119	0.127	0.103	0.034	0.039	0.052	0.030
2East-1	ND	0.030	0.058	0.046	0.013	0.016	0.018	0.012
2East-2	ND	0.035	0.053	0.046	0.010	0.024	0.017	0.016
West-1	ND	ND	ND	ND	0.034	0.023	0.010	0.016
West-2	ND	ND	ND	ND	0.034	0.023	0.014	0.017

[a] INTERVAL 1 = background levels 2/18/92; INTERVAL 2 = during application 2/20/92 from 12:00 to 13:15; INTERVAL 3 = 2/20/92 from 13:15 to 15:15; INTERVAL 4 = 2/20/92 from 15:15 to 20:15; INTERVAL 5 = 2/20-21/92 from 20:15 to 06:30; INTERVAL 6 = 2/21/92 from 06:30 to 13:15; INTERVAL 7 = 2/21-22/92 from 13:15 to 13:15; INTERVAL 8 = 2/22-23/92 from 13:15 to 13:15

[b] not detected; minimum detection limit is 0.004 ug/m^3 for a 4 hour sample

Source: Kollman 1995

It is assumed that since formulations of Bravo contain 19% by volume of chlorothalonil, hexachlorobenzene, which as a contaminant is in the 54% by volume inert ingredients category, could also be detected at the levels set out in Table 5.3. Table 5.3 illustrates that for every single spray of pesticide containing chlorothalonil, there are at least three days of exposure to chlorothalonil residues left in the air. Since there are no comparable documents for cypermethrin and ethylenethiourea, it is assumed that three days of exposure to these chemicals per spray also applies. The actual residues in the air that applicators and others are exposed to could be more or less concentrated and could dissipate more or less quickly.

5.2 THE DOSE-RESPONSE FUNCTION

Concentration levels for this thesis study were estimated on the basis of the maximum, middle high average, average, middle low average and minimum of the information in Table 5.3 to give concentration rates of 0.158, 0.066708, 0.033354, 0.016677 and zero ug/m^3 respectively[11], per one application of spray for three consecutive days. To convert these concentrations into doses, the following equation (3) from Chapter 2 was used:

$$ED = \frac{CR * C * EF}{BW}$$

where ED is the estimated dose, expressed as milligrams of the contaminant inhaled per kilogram of body weight per day. This is what is being calculated;

CR is the ingestion and/or inhalation rate. Since the dose-response function is based on inhalation exposures, a rate of 20m^3 of air breathed per day, averaged over a lifetime of 70 years is used. Additionally, it is assumed that the affected population is breathing the concentrations at a constant level over a 24 hour period;

C is the exposure concentration. This is listed above in units of ug/m^3 and converted to mg/m^3 for the calculation;

EF is an exposure factor. This number will be the portion of days per year that the farmer population is exposed to pesticide residues in the air over a lifetime of 70 years; and

BW is body mass in kilograms. A standard weight of 60 kilograms is used.

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[11] ND values in Table 5.3 were taken as zeroes in the calculation of averages.

The ED is then taken as the estimated daily intake (EDI)[12] and multiplied by the slope factor of hexachlorobenze (1.6 [IRIS 1998]), which gives the level of risk at the specified dose. Table 5.4 shows the EDI values calculated using equation (3) and the levels of risk at those exposures.

Table 5.4: Estimated daily intakes (EDI) and subsequent risk estimates for chosen concentration rates (in mg/m^3) under Conventional/NewLeaf/NewLeaf Plus and NewLeaf Pro growing practices

Concentrations	Conventional/NewLeaf/NewLeaf Plus Growing Practices[a]			NewLeaf Pro Growing Practices		
	EDI	Risk	1/x[b]	EDI	Risk	1/x
0	0	0		0	0	
1.6677E-5	5.9E-7	9.5E-7	1052718	1.8E-7	2.9E-7	3417334
3.3354E-5	1.19E-6	1.9E-6	526359	3.7E-7	5.9E-7	1708667
6.6708E-5	2.37E-6	3.8E-6	263180	7.3E-7	1.17E-6	854334
1.58E-4	5.62E-6	9.0E-6	111115	1.73E-6	2.77E-6	360702

[a] Due to the difference in the number of pesticide applications containing identified active ingredients that are applied under each growing practice, the exposure factor changes, changing the results of the dose the population is exposed to and the subsequent risk associated with the exposure.

[b] 1/x represents (e.g.) one person in 111,115 will contract cancer at the EDI of 5.62E-6.

The risk estimates given in Table 5.4 were then used to calculate estimates of the monetary value of nonfatal and fatal cancer that each exposed individual is expected to be willing to pay to not apply the pesticides. The risk estimates were combined with values

of illness taken from the Royal Society Publication "AQVM Companion Manual to the Report of an Expert Panel to Review the Socio-Economic Models and Related Components Supporting the Development of Canada-Wide Standards for Particulate Matter and Ozone" [Stratus Consulting 2001]. These values take into account the average of the estimates for those both under and over 65 years of age, since as discussed in Section 2.1.3, the value that a 75 year old places on life is different from that expressed by a 25 year old. Additionally, the values of cancer were those listed as fatal/non-fatal non-specific cancer cases resulting from exposure to Acetaldehyde, 1,3 Butadienne, and Formaldehyde. The other values listed in Stratus Consulting (2001) were for a specific fatal form of leukemia after exposure to benzene.

Table 5.5: Nonfatal and fatal cancer illnesses of a non-specific form and their values as reported in Stratus Consulting (2001)

Illness	Value of Illness
Nonfatal Cancer Case (non-specific)	$305 000.00
Fatal Cancer Case (non-specific)	$4 300 000.00

The risk estimates were also combined with the survival and death rates of liver, lung and thyroid cancer as shown in equations (9) and (10). This rate was calculated as the average survival rate for these cancers, expressed as a percentage, or 38.3% for the survival rate and 61.7% for the death rate [Gloeckler Ries 1994]. By employing a form of benefits transfer using the monetary values published in AQVM Companion Manual

[12] The EDI normally includes the estimated doses from all routes of exposure; however, since this thesis study only takes into account exposure through inhalation, the ED takes the place of the EDI in this calculation.

[Stratus Consulting 2001], the annual monetary value to PEI potato farmers of fatal and nonfatal cancers was calculated using equations (9) and (10) below:

Annual Value of Nonfatal Cancer = \$305,000.00 * Risk * Survival Rate (9)

Annual Value of Fatal Cancer = \$4,300,000.00 * Risk * Death Rate (10)

where risk values are as calculated and presented in Table 5.4; the survival rate is an average survival rate for liver, lung and thyroid cancer, expressed as a percentage; and the death rate is 100 - survival rate, expressed as a percentage.

Further assumptions are applied to estimate the total health costs for PEI farmers. Health effects are estimated for the applicator, in this case the farmer, and their families only. There are 650 potato farmers in PEI, and an average family size of 4 people is assumed. This estimation further assumes that all 4 people in the farm family, but no one else, is exposed to identical doses of the chemical after it has been sprayed and absorb the chemical at the same rate.

Relative to the possible effect of application of chemicals from alternate sources, it is assumed that there is no drift of the chemicals. Further, it is assumed that residues on food are zero/undetectable for all pesticide active ingredients. Finally, each spray application is assumed to be applied at least three days apart so that there is no overlap between applications. That is, each spray application is taken to be equal to three days of exposure, and it is assumed that the dose absorbed by the affected individuals throughout this three day time period is constant.

CHAPTER 6: HEALTH EFFECTS: RESULTS AND DISCUSSION

6.1 RESULTS

The results section is organized as follows. First, a table is presented detailing the annualized monetary costs of illness found using the spraying schedule recommended on the pesticide labels. This table was developed using the assumptions and equations contained in Chapter 5, Section 5.2 and illustrates the annual health costs to PEI farmer applicators and their families of using the dangerous identified chemicals. Second, a comparison will be made between the annual health estimates found under the conventional spraying schedule and the NewLeaf Pro spraying schedule. Third, the results from the partial budget will be discussed as they pertain to land values. Next, the wider impact of pesticide drift will be discussed in the context of the entire population of PEI. Finally, the consequences of using fewer pesticides will be discussed in relation to the environment.

Table 6.1: Annualized costs of illness using spraying schedule recommended by provincial Potato Specialist and frequency of pesticide sprays under Conventional, NewLeaf, NewLeaf Plus and NewLeaf Pro growing practices (Table 4.2) for the affected farmer population of Prince Edward Island

			Concentration Rates in mg/m^3 for Conventional, NewLeaf and NewLeaf Plus potato varieties[a]				Concentration Rates in mg/m^3 for NewLeaf Pro potato variety[b]			
Illness	Cost of Illness	%[c]	1.6677E-5	3.3354E-5	6.6708E-5	1.58E-4	1.6677E-5	3.3354E-5	6.6708E-5	1.58E-4
Nonfatal Cancer	$305,000.00	38.3	$0.11	$0.22	$0.44	$1.05	$0.03	$0.07	$0.14	$0.33
Fatal Cancer	$4,300,000.00	61.7	$2.52	$5.04	$10.08	$23.88	$0.78	$1.55	$3.11	$7.36
Total Cost per applicator			$2.63	$5.26	$10.52	$24.93	$0.81	$1.62	$3.25	$7.69
Total Cost for Affected Population[d]			$6,838.00	$13,676.00	$27,352.00	$64,818.00	$2,106.00	$4,212.00	$8,450.00	$19,994.00
Total Health Savings realized when growing NewLeaf Pro potatoes			$4,732.00	$9,464.00	$18,902.00	$44,824.00				

[a] Exposed to hexachlorobenzene, cypermethrin and ethylenethiourea for 39 days per year (1 spray Ripcord, 4 sprays Bravo, 8 sprays Dithane = 13 sprays * 3 days exposure/spray)

[b] Exposed to hexachlorobenzene, cypermethrin and ethylenethiourea for 12 days per year (1 spray Ripcord, 3 sprays Dithane = 4 sprays * 3 days exposure/spray)

[c] Taken as an average of survival/death rates for liver, lung and thyroid cancers

[d] Affected population refers to each of the 650 potato farmers and their family (assumes 4 people in a family unit), or 2600 people

To understand the results presented in Table 6.1, some assumptions must be reiterated. The first is that exposure to hexachlorobenzene, cypermethrin, and ethylenethiourea are represented by the recommended spraying schedule set out by the provincial potato specialist for Conventional, NewLeaf, NewLeaf Plus and NewLeaf Pro potatoes. Second, even though the total number of pesticides sprayed decreased when using NewLeaf and NewLeaf Plus potatoes, the frequency of spraying pesticides containing the afore mentioned chemicals did not change. This is because the chemicals in question are used for controlling corn borer and early/late blight. The NewLeaf potatoes were modified for control of the Colorado potato beetle; NewLeaf Plus potatoes were modified for resistance to Colorado potato beetle and potato leafroll virus; while NewLeaf Pro potatoes were in developmental stages for the control of Colorado potato beetle, potato leafroll virus, potato virus Y, and late blight.

Third, the spraying schedule set out for NewLeaf Pro potatoes was based on the potato specialist's knowledge and expectations of the expected results of growing this "in developmental stages" potato variety. Since the potato was never developed or tested, this component of the spraying schedule, as well as the results gleaned from it, are 'best guess' results.

Finally, it should be noted that the spraying schedule that was set out in Table 4.2 would probably not be used in consecutive years, due partly to changes in the pest and

disease circumstances, which are in great part a product of the weather[13], and partly to resistance of pests and viruses[14]. The spraying schedule is meant to illustrate a "typical" spraying schedule that is based on the current popular pests, viruses and diseases in PEI potato production.

The final results are the estimates of the annual monetary value of health effects from using pesticides containing the identified dangerous chemical active ingredients of hexachlorobenzene, ethylenethiourea, and cypermethrin. These are presented in Table 6.1 and indicate that the annualized cost, in monetary terms, of the health effects for the assumed affected population from growing Conventional potatoes versus growing NewLeaf Pro potatoes ranges between $4,732.00 and $44,824.00, depending on the level of exposure to which the affected population is subjected. It should be noted that, in this section when the word "Conventional" is used, it refers to Conventional, NewLeaf and NewLeaf Plus potatoes, as the number of sprays containing the identified dangerous active ingredients for these varieties are the same. This occurs because the dangerous chemicals are used to control corn borer and early/late blight. These numbers are not overly large, in that they represent a per person annualized cost of $1.82 to $17.24 when Conventional potatoes are grown instead of NewLeaf Pro potatoes.

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[13] The weather could provide a perfect breeding ground for late blight one year, requiring numerous sprays, whereas in the following year, there could be no signs indicative of the disease, and sprays could be just for basic protection.

[14] Pests and viruses have been shown to be mutating to the extent that a pesticide that controlled their spread five years ago will not work today. Some examples of this are Potato Virus Y and the Colorado Potato Beetle [MacPhail 2003, Coffin 2003].

To convert these numbers to an average health cost per hectare, the individual monetary estimates were divided by the average number of hectares that each potato grower farms. That is,

Health cost per hectare = Cost/farmer * 2600 farmers/56000 hectares (11)

The total health costs accrued from growing Conventional, NewLeaf and NewLeaf Plus potatoes ranges from 3¢ to 27¢ per hectare (depending on level of exposure), but the health costs accrued from growing NewLeaf Pro potatoes range from 1¢ to 8¢ per hectare. Assuming the maximum exposure levels apply (and therefore the maximum monetary costs of illness), the benefit to health per hectare that comes from growing NewLeaf Pro potatoes is 19¢. The previous partial budget saving estimates of Table 4.5, which considered pesticide application cost saving alone, are modified in Table 6.2 to encompass both the pesticide savings and reduced health costs of growing genetically modified potatoes:

Table 6.2: Per hectare savings overall from growing genetically modified potatoes as compared to growing conventional potatoes

	Conventional	NewLeaf	NewLeaf Plus	NewLeaf Pro
Pesticide Savings	$0	$118.57	$207.28	$645.71
Health Savings[a]	$0	$0	$0	$0.19
Total Savings	$0	$118.57	$207.28	$645.90

[a] Health savings are for the maximum exposure level, with the only savings discovered when NewLeaf Pro potatoes are grown, due to the decrease in spray frequency of identified dangerous chemicals.

[b] Pesticide savings encompass the chemical and non-chemical application costs, as well as the royalty fees Monsanto charges for the use of its seed.

It can be seen from Table 6.2 that the health benefits of less pesticide use, as estimated, are much smaller than the financial savings from reduced pesticide use.

The arguments against growing genetically modified crops are the topic of much discussion in the literature and encompass issues of public perception, risk assessment, as well as ethical and environmental considerations [see Goga et.al. 2002, Krimsky 1997]. The purpose of this study was not to discount the arguments made against the genetic modification of crops for pest and disease resistance, but merely to point out its findings. These are that there are small potential savings in health costs associated with pesticide reduction and significant savings to the farmer applicator due to reduced pesticide usage associated with growing genetically modified potato varieties in a major potato growing region of Canada.

The most significant savings to PEI farmer applicators are found to apply when growing NewLeaf Pro potatoes, which gives estimated aggregate savings of $36,170,400.00. These potatoes might or might not perform as assumed. However, there is the possibility that they could have exceeded expectations and outperformed the original assumptions or that Monsanto could have improved upon the technology and created another modified potato that outperformed the expected performance of NewLeaf Pro potatoes. The annualized savings from the health costs resulting from using NewLeaf Pro potatoes is a maximum of $44,824.00, which is only 0.12% of the total savings, whereas the annualized savings from pesticide reduction is 99.88% of the total savings. When compared to the savings realized when growing NewLeaf potatoes

($6,639,920.00) and NewLeaf Plus potatoes ($11,607,680.00), the annual savings are more than tripled when NewLeaf Pro potatoes are grown.

Savings of the extent documented could be expected to have major implications on land values in PEI. Currently, an acre of prime potato growing land sells for $3,000.00 [MacPhail 2004]. A question then becomes, "Are land values affected by the anticipated stream of net benefits?" Economic theory suggests that the stream of anticipated net benefits will be discounted back into the present value of the land.

The appropriate discount rate in this instance should at least reflect the market interest rate. The Farm Credit Canada information hotline indicated that a 10 year fixed interest rate of 7.2% is available for farm loans (February 2004). This encompasses the cost of borrowing funds to purchase a tract of land but does not reflect opportunity costs and risk issues associated with such an investment analysis.

Opportunity costs involve the best alternate return that could have been achieved with that sum of money invested in the land. For instance, if the person in question is already a potato farmer, expenditure to acquire new land could have been spent on additional farm equipment or on implementing alternative pest treatment programs. In the case of risk, at this point in time, potato sales are lower and inventories higher than normal, a feature attributed to the Atkins' diet and an overproduction of potatoes [Morris 2004]. [This diet eliminates the majority of carbohydrates from the diet in order for the users of the program to lose weight, which in almost all cases means that potatoes are completely eliminated from a person's diet.] For these reasons and the scope of this study, the rate of 10% will be used to capture both the opportunity costs and the increased

risk of potato production at this point in time.

To calculate the savings in perpetuity, equation (12) is used.

$$\frac{AV}{[(1+r)^3-1]} \qquad (12)$$

where AV is the annual value of the savings, r is the discount rate, with consideration being given to the fact that potatoes can only be grown on the same parcel of land once every three years due to crop rotation practices. Implicit assumptions are also made that the savings from reduced chemical use as presented in Table 6.3 are maintained over time and would continue to apply each year and that the payment for seed, representing the annual right for farmers to use the innovation, would not change.

Table 6.3: Discounted net benefits per hectare of reduced pesticide application in perpetuity

Potato	Annual Savings from Reduced Chemical Use	Increase in Land Values
NewLeaf	$118.57	$358.22
NewLeaf Plus	$207.28	$626.22
NewLeaf Pro	$645.71	$1950.79

From a conversion of land values from $3,000.00 per acre to $7,410.00 per hectare, it can be seen that if the savings from reduced pesticide usage were to be fully capitalized into land values, land value could increase by amounts between $350 and $1950 per hectare, depending on the type of genetically modified potato grown.

The contribution to total operating costs per hectare provided by chemical inputs is estimated to be between 10 and 15% [MacDonald 1997]. Chemicals inputs represent approximately 25% of the input costs that the farmer faces each year (excluding wages,

licensing fees, custom work, repairs, taxes and insurance), with fertilizer and seed each representing an additional 25% of input costs, based on average cost estimates for 10 potato farms for the 1994 and 1995 crop years [MacDonald 1997]. It should be noted that in any year chemicals would represent a smaller or larger portion of costs depending on the pest, virus and disease problems faced by the farmer, as well as the farmers' individual tolerance of the problem in the field [MacPhail 2003].

6.2 IMPACT OF PESTICIDE DRIFT ON HEALTH COSTS

The potential drift of the chemicals sprayed, which raises the possibility of health effects on other community members, was not taken into consideration in estimates presented to this point in the study. However, from a study conducted for the Government of California[15], there is a clear indication that air samples taken in a 50 to 70 km radius circle from the spraying field contained traces of the active pesticide ingredients. In that study, air concentrations from three locations were tested for one of 17 identified chemicals, one of which was chlorothalonil. These drift residues were measured in ng/m^3 and their presence was concluded to be partially dependent on wind speed and wind direction. The mean values of the air concentrations for chlorothalonil for the three field locations in the study were 14.74, 2.11 and 2.78 ng/m^3 for the south wind direction and 23.27, 6.57, and 13.44 ng/m^3 for the north wind direction. Converting

[15] See Majewski and Baston 2002.

these measurements into ug/m^3, they become 0.01474, 0.00211 and 0.00278 ug/m^3 and 0.02327, 0.00657, and 0.01344 ug/m^3 respectively.

Based on assumptions presented in the previous chapter, it is assumed that testing for hexachlorobenzene drift would give similar results as those found in California for chlorothalonil. The air concentrations are all clearly above the 0.002 ug/m^3 minimum listed in the dose response function for hexachlorobenzene, and therefore would carry with them a greater than 1 in a million risk of contracting cancer based on lifetime exposure. Prince Edward Island is a small island and potato fields normally extend up to property edges, with no buffer zone between houses and the fields. Due to the distance that the drift can travel, and the distribution of farms on the land base of Prince Edward Island, it could be assumed that every person within a 50 km radius of where the pesticide is sprayed will be exposed to the chemical active ingredient of the pesticide, although the time frame of exposure is not known. Using the assumptions presented in the previous paragraph and assuming ethylenethiourea and cypermethrin drift would be found in similar concentrations, this suggests that each person in Prince Edward Island is potentially exposed to a minimum of 13 days per year at risks greater than 1 in a million of contracting cancer over his or her lifetime, or for as long as they live on the Island when conventional potatoes, NewLeaf potatoes, or NewLeaf Plus potatoes are grown. This would significantly increase the total health cost estimates presented in Table 6.1, and in turn becomes a policy issue to be explored in further detail at a later date.

In contrast, if the NewLeaf Pro potatoes were grown and the spraying schedule was as outlined in Chapter 4 (Table 4.2), the population of Prince Edward Island would

potentially be exposed to a minimum of 4 days at the same air concentrations, reducing the total health costs to the province by approximately 70%, as illustrated in Table 6.4.

Table 6.4: Annualized costs of illness using spraying schedule recommended by the provincial Potato Specialist and frequency of pesticide sprays under Conventional, NewLeaf, NewLeaf Plus and NewLeaf Pro growing practices(Table 4.2) for the entire population of Prince Edward Island

			Concentration Rates in mg/m^3 (13 days per year)[a]				Concentration Rates in mg/m^3 (4 days per year)[b]			
Illness	Cost of Illness	%	1.6677E-5	3.3354E-5	6.6708E-5	1.58E-4	1.6677E-5	3.3354E-5	6.6708E-5	1.58E-4
Nonfatal Cancer	$305,000.00	38.3	$0.04	$0.07	$0.15	$0.35	$0.01	$0.02	$0.05	$0.11
Fatal Cancer	$4,300,000.00	61.7	$0.84	$1.68	$3.36	$7.96	$0.26	$0.52	$1.04	$2.46
Total Cost per Person			$0.88	$1.75	$3.51	$8.31	$0.27	$0.54	$1.09	$2.57
Total Cost for Farmer Population[c]			6,838.00	13,676.00	27,352.00	64,818.00	2,106.00	4,212.00	8,450.00	19,994.00
Total Cost for Remaining PEI Population[d]			119,100.08	236,846.75	475,046.91	1,124,683.71	36,542.07	73,084.14	147,521.69	347,826.37
Total Health Costs			125,938.08	250,522.75	502,398.91	1,189,501.71	38,648.07	77,296.14	155,971.69	367,820.37
Total Health Savings realized when growing NewLeaf Pro potatoes			$87,290.01	$173,226.61	$346,427.22	$821,681.34				

[a] The monetary values of health costs are applicable when Conventional, NewLeaf and NewLeaf Plus potato varieties are grown.

[b] The monetary values of health costs are applicable when NewLeaf Pro potato varieties are grown. Note that this potato was in promising stages of development, but never tested or grown. The values expressed in this section are based on the best guess spraying schedule presented in Table 4.2.

[c] Farmer Population is equal to 2600 people (650 farmers and 4 people per farm family). Monetary values for this section are taken from Table 6.1 and express the fact that this population is exposed to 3 days of chemical residues per spray.

[d] PEI Population is equal to 137 941as per Statistics Canada October 1, 2003 preliminary demographic statistics, less 2600 farmer population http://www.statcan.ca/Daily/English/031218/d031218c.htm These monetary values reflect the fact that the remaining population is only exposed to one day of chemical residues per spray.

The estimates in Table 6.4 are derived using PEI-specific information, however, in potato intensive areas with larger populations, such as New Brunswick, or in the United States in the potato growing regions of Wisconsin, Idaho, Washington, and North Dakota where the population is larger, there potentially could be larger aggregate health costs from pesticide drift than found in this study for PEI.

6.3 THE ENVIRONMENT

This study did not encompass the environmental consequences that are associated with pesticide application or the possible benefits associated with a reduction in pesticides. There are many aspects that could be considered, including potential residues or contamination of ground water and well water, soil residues, effects on birds, and effects on aquatic animals. Each individual pesticide sprayed could have effects in each of the above-mentioned areas.

6.3.1 Resistance of target weeds to pesticides

A major concern for all farmers, industry and environmental groups is the issue of externalities that can be associated with the use of pesticides in agriculture. In a study by Leguizamon (2001), it was concluded that overuse of glyphosate, which is one of the active ingredients found in two of the pesticides used on Prince Edward Island, would result in "changes in weed flora (selection for species with extended germination pattern and/or tolerances), decrease in community species richness and diversity, and increased risk of outbreaks of resistant weeds." These types of conclusions are likely to be

applicable not only to herbicides, but to insecticides and fungicides as well. The use of genetically modified crops with resistance built into the actual plant could limit these impacts and possible off-site effects that may be associated in periods of heavy rainfall when it is feared that chemicals may wash into adjacent waterways.

6.3.2 Fish Kills

During the past three years, instances when fish kills in PEI have been observed (which typically occur when pesticide applications are followed by heavy rainstorms) have dramatically increased. From 1990 to July 2002 there were 18 reported fish kills, of which farm-chemical runoffs have been viewed as contributory factors in all cases except one [Freeze 2002]. During the summer of 2002, it was intimated that each time there was enough precipitation to wash silt into a watercourse, toxins were carried in the silt that killed aquatic wildlife [O'Connell 2002]. The dead fish count reported by Freeze (2002) was well over 10,000 in the first weeks of July in the Wilmot area, and no data have yet been reported about amphibians or insects that may also have been killed by the toxins in the water. This is not the only environmental effect being noticed – another that has been observed (but not as studied or documented) is the reduction in the number and types of birds in Prince Edward Island. The PEI government has agreed to take preventative measures, in the form of creating agricultural buffer zones and passing crop-rotation laws to reduce the problem [Info PEI 2002].

The reported fish kills are not the only apparent environmental consequences of using pesticides; the problem also appears to affect PEI's recreational fishing industry. In a 1999 report released by the Atlantic Salmon Federation and quoted in Freeze (2002), it

is stated that "up to four million tonnes of soil end up in streams, along with chemical residues".

6.3.3 Inert Ingredients

Pesticide manufacturers are not required by law to tell users which inert ingredients are contained in their pesticide formulations. This is partially because these inert ingredients are considered trade secrets. It should be noted that in some cases, the inert ingredients are more toxic to human health and the environment than the actual active ingredient used for pest or disease control [Journal of Pesticide Reform 1999]. An example of this is hexachlorobenzene, an inert ingredient found in chlorothalonil formulations, such as Bravo. This study has concentrated on the effects that hexachlorobenzene could have on human health at different exposure rates, but it is unknown how much hexachlorobenzene is actually in the Bravo formulation, only that it is contained in the 54% by volume inert ingredient category.

6.4 SUMMARY

Health costs associated with chemical use on PEI (0.12% of total savings) are not as appreciable a cost when compared to the savings from chemical inputs (99.88% of total savings) when NewLeaf Pro potatoes are grown. Land values have the potential to increase by $500 to $3000 per hectare based solely on the savings from reduced chemical use of growing the genetically modified potatoes.

The issue of pesticide drift indicates that there could potentially be higher health costs than originally accounted for, but that these costs are still not as significant as the savings found from reduced chemical use. The environmental consequences of chemical use are outlined, although they should be quantified to give a better overall view of the costs of using pesticides.

CHAPTER 7: CONCLUSIONS AND LIMITATIONS

7.1 MODEL SUMMARY

This study used a partial budget (in Chapter 4) to estimate the savings from the reduced use of chemicals found when growing genetically modified potatoes. This study also used a dose response function for hexachlorobenzene (in Chapter 5) and applied this to two other EPA "probable/possible carcinogens" classified chemicals, all of which were either active or inert ingredients or a metabolic by-product of pesticide formulations used in potato production in Prince Edward Island. By applying health risk assessment techniques, the level of risk of contracting cancer over a lifetime of exposure was examined for various concentration levels. By combining this function with health cost information derived through Value of Statistical Life techniques, estimates of health costs of cancer risks aggregated over each PEI farmer applicator, as well as their families, were calculated.

7.2 SUMMARY OF RESULTS

Using the assumptions set out in Chapter 5, the results indicate that the annualized monetary health costs for the assumed affected population of farmer applicators and their families that arise from the inability to reduce applications of identified dangerous pesticides through Conventional, NewLeaf, and NewLeaf Plus growing practices ranges from $4,732.00 to $44,824.00. These estimates are based on scenarios in which farmers

grow NewLeaf Pro potatoes as opposed to Conventional potatoes. The estimates depend on the level of exposure to which the affected population is subjected.

The chances of accidental exposure are further lessened by safe handling practices, due to education awareness programs and courses that must be taken in order to handle and apply pesticides. It is assumed that these measures result in the affected population being exposed only to the residues of chemical remaining in the air following application.

The lack of knowledge and availability of information relative to safe pesticide use in developing countries, as discussed in Chapter 2, may lead to different conclusions in that environment if there are higher concentrations of residues after applications of pesticide because of lack of knowledge regarding proper handling, spraying and storage procedures. In this case, the potentially higher yields that may be attainable using pesticides could be less appealing to the farmers of developing countries than growing genetically modified crops.

While the health costs of contracting cancer over a lifetime of exposure are quantifiable, they are not as significant as one might have thought. This is especially true when health costs are compared to the savings that farmers would experience when growing genetically modified potatoes due to the reduction in chemical inputs. These savings range from $6 million to $36 million per year, depending on the type of genetically modified potato grown.

7.3 LIMITATIONS AND FURTHER RESEARCH

The dose response function is the only available information at this time to link levels of chemical inhalation exposure to increases in cancer risks, and is only available for a limited number of chemicals. To that end, one of the two main limitations of this study is the assumption that the chemicals ethylenethiourea and cypermethrin have the same toxicity as hexachlorobenzene and would have a similar dose response function. Another major limitation is the assumption that hexachlorobenzene, ethylenethiourea and cypermethrin residues would behave as those of chlorothalonil in respect to the level and persistence of chemical concentrations remaining after time. The limitations of these two key pieces of information should be recognized, as results gained from the use of actual studies could change when compared to the results gained in this study.

Another limitation is that NewLeaf Pro potatoes, although in a promising developing stage, were not completed or grown. The spraying schedule that was developed in regards to NewLeaf Pro potatoes was developed using the PEI provincial potato specialist's knowledge and best guesses. The results reported here could change depending on the success that Monsanto had in the development of the new potatoes and the strength/capability of the modified resistances.

Other limitations of the study are that it was not possible to study environmental consequences of pesticide use in any detail. Instances of fish kills were mentioned in Chapter 6. A more comprehensive assessment would assess the economic impact that fewer pesticide sprayings could have on the PEI environment.

Pesticide drift has long been a topic of concern for environmental groups and others, including government. In Chapter 6, a discussion was presented using California drift data of chlorothalonil that indicates that every person in a 50 to 70 km radius of the pesticide application would be exposed to quantifiable levels of the chemical. Use of the following two assumptions resulted in a conclusion that the entire population of PEI could be at an increased risk of contracting cancer over a lifetime of exposure. First, that exposure to chlorothalonil is in the same concentrations as exposure to its' pesticide formulation inert ingredient hexaclorobenzene. Second, ethylenethiourea and cypermethrin were detectable in the same concentrations as chlorothalonil and had the same toxicity as hexachlorobenzene. This argument could be carried further to encompass more populated areas of intensive potato farming such as New Brunswick, Idaho, or any other of the major potato producing states in the United States.

Finally, it has been found in studies summarized by the Extension Toxicology Network [EXTOXNET][16] that exposure to active ingredients can cause bronchitis, respiratory problems, cardiac problems, asthma, and flu-like symptoms if an individual is overexposed to the chemicals. These illnesses are not taken into account in this study because as yet research has not been undertaken that can link a specific rate of chemical exposure to these problems.

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[16] See http://pmep.cce.cornell.edu

Further research that could be done on this topic will depend on toxicologic or epidemiological studies of the actual dose response functions (as defined by the EPA) of probable human carcinogenic chemicals, as well as the leftover residues after a spray of pesticide, for the several different active ingredients prominent in potato production. These two pieces of information would result in much more precise estimates of the health costs incurred by potato farmers from pesticide use. The environmental costs should also be assessed in more detail to give a more comprehensive view overall of the costs of using pesticides in modern agriculture.

7.4 FINAL REMARKS

The savings from reduced chemical use presented in the partial budget in Chapter 4 are fairly straightforward, however, due to lack of epidemiological and air residue concentration information, the results and inferences made from the knowledge and assumptions contained in this paper in regards to health costs may vary quite substantially from real life. There needs to be a call for more studies of this nature, in developed and developing countries, to help identify where the biggest effects from GM technologies might be. These studies will aid in future policy decisions and consumer education and awareness programs in relation to the costs and benefits of both pesticide use and genetically modified crops.

REFERENCES

Alberta Report. 1998. "New biotech spud is a superior French fryer." *Alberta Report.* 24(43): 19.

Arseneau, Walter. 2002. Personal communication.

Athayde, Marcella. Nov/Dec 2001. "Monsanto Drops Its Biotech Potato." *World Watch.* 14(6): 5.

Balasubramaniam, A. 1997. "Control of pesticides in certain countries of south-east Asia." *Environmental behaviour of crop protection chemicals: proceedings of an international symposium on the use of nuclear and related techniques for studying environmental behaviour of crop protection chemicals.* Vienna, Austria. 1-5 July 1996: 61-71.

Blomquist, Glenn C. 2000. "Economics of Value of Life." *International Encyclopedia of the Social and Behavioral Sciences.* 24:16132-9.

Boswall, Peter. 1998. "Prince Edward Island Potatoes." AGDEX 163/00.

Byfield, Mike. December1999. "Greenpeace mashes modified potatoes." *Report Newsmagazine (BC Edition).* 26(44): 23.

CASRN [Chemical Abstracts Service Registry Number] 96-45-7. "Ethylene Thiourea." *Available Online: Http://toxnet.nlm.nih.gov/cgi-bin/sis/search/f?./temp/~AAA36aiCC:1* .

CEPA [Canadian Environmental Protection Agency]. 2004. "NPRI-listed, CEPA-toxic and Carcinogenic Pollutants in Canada." *Available Online: Http:// www.ec.gc.ca/pdb/npri/2002Highlights/NPRI2000Overview/4pollutants_e.cfm* .

CFIA [Canadian Food Inspection Agency]. 1996. "Decision Document DD96-06: Determination of Environmental Safety of NatureMark Potatoes' Colorado Potato Beetle (CPB) Resistant Potato (*Solanum tuberosum* L.)." *Available Online: http://www.inspection.gc.ca/english/plaveg/bio/dd/dd9606e.shtml*

Coffin, Robert. 2003. Personal Communication.

Cox, Caroline. 1997. "Chlorothalonil: Fungicide Factsheet." *Journal of Pesticide Reform*. 17(4): 14-20.

Cox, Caroline. 2001. "Imidacloprid: Insecticide Factsheet." *Journal of Pesticide Reform.* 21(1):15-21.

Dyro, Frances. 2001. "Organophosphates." *Available Online:* *Http://www.emedicine.com/neuro/topic286.htm* .

EXTOXNET [Extension Toxicology Network]. 1993a. "Cypermethrin." *Pesticide Information Profiles. Available Online:* *http://pmep.cce.cornell.edu/profiles/extoxnet/carbaryl-dicrotophos/cypermet-ext.html* .

EXTOXNET [Extension Toxicology Network]. 1993b. "Permethrin." *Pesticide Information Profiles. Available Online:* *http://pmep.cce.cornell.edu/profiles/extoxnet/metiram-propoxur/permethrin-ext.html* .

EXTOXNET [Extension Toxicology Network]. 1994. "Pyrethrins and Pyrethroids." *Pesticide Information Profiles. Available Online:* *Http://ace.ace.orst.edu/info/extoxnet/pips/pyrethri.htm* .

EXTOXNET [Extension Toxicology Network]. 1995. "Deltamethrin." *Pesticide Information Profiles. Available Online:* *http://pmep.cce.cornell.edu/profiles/extoxnet/carbaryl-dicrotophos/deltamethrin-ext.html*.

EXTOXNET [Extension Toxicology Network]. 1996a. "Carbofuran." *Pesticide Information Profiles. Available Online:* *Http://extoxnet.orst.edu/pips/carbofur.htm* .

EXTOXNET [Extension Toxicology Network]. 1996b. "Imidacloprid." *Pesticide Information Profiles. Available Online:* *Http://ace.ace.orst.edu/info/extoxnet/pips/imidaclo.htm* .

EXTOXNET [Extension Toxicology Network]. 1996c. "Mancozeb." Pesticide Information Profiles. *Available Online:* *http://pmep.cce.cornell.edu/profiles/extoxnet/haloxyfop-methylparathion/mancozeb-ext.html* .

Farm Credit Canada. 2004. "Farm Credit Canada Information Hotline."

Freeze, Colin. July 17, 2002. "Death by Potato." *Globe and Mail. Available Online:* *Http://www.eco-vie.com/ENN_PEI_FishKill.html* .

Gloeckler Ries, Lynn A. 1994. "Cancer Survival Rates: Changes in the 5-Year Survival Rates by Primary Cancer Site, Whites." *Available Online: http://seer.cancer.gov/publications/raterisk/rates29/html* .

Goga, B., F. Clementi & R. Chadwick. 2002. "Safety assurance of foods: risk management depends on good science but it is not a scientific activity." *Journal of Agricultural and Environmental Ethics*. 15(3): 305-313.

Government of Prince Edward Island. 2002. "28th Annual Statistical Review 2001." *Available Online: Http://www.gov.pe.ca/photos/original/28annualreview.pdf* .

Hallman, William K. & Helen L. Aquino. 2003. Consumer Perceptions of Genetically Modified Food. *Rutgers University: New Jersey.*

Health Canada. 1995. "Investigating Human Exposure to Contaminants in the Environment: A Community Handbook." Cat. No. H49-96/2-1995E. *Available Online: Http://dsp-psd.communication.gc.ca/Collection/H49-96-2-1995E-1.pdf* .

Health Canada. 1999a. "Colorado Potato Beetle and Potato Leaf Roll Virus Resistant Potato Lines RBMT21-129, RBMT21-350, RBMT22-082. FD/OFB-099-127-B." *Available Online: Http://www.hc-sc.gc.ca/food-aliment/mh-dm/ofb-bba/nfi-ani/e_ofb-099-127-b.html* .

Health Canada. 1999b. "Colorado Potato Beetle and Potato Virus Y Resistant Potato Lines SEMT15-02, SEMT15-15, RBMT15-101. FD/OFB-099-127-A." *Available Online: Http://www.hc-sc.gc.ca/food-aliment/mh-dm/ofb-bba/nfi-ani/e_ofb-099-127-a.html* .

Health Canada Food Directorate. *Available Online: Http://www.hc-sc.gov.ca* .

HIARC [Hazard Identification Assessment Review Committee]. 1998. "Hazard ID Committee Report." *Available Online: Http://www.epa.gov/pesticides/op/hazidrpt.pdf* .

Hossain, Ferdaus, Benjamin Onyango, Adesoji Adelaja, Brian Schilling & William Hallman. 2002. "Consumer Acceptance of Food Biotechnology: Willingness to Buy Genetically Modified Food Products." *Rutgers University Working Paper: New Jersey.*

IARC [International Agency for Research on Cancer]. 2004. "Overall Evaluations of Carcinogenicity to Humans." *Available Online: Http://www.iarc.fr/* .

Info PEI. 2002. "Watercourse Buffer Zones." *Available Online: http://www.gov.pe.ca/infopei/onelisting.php3?number=61371* .

IRIS [Integrated Risk Information System]. 1998. "Hexachlorobenzene (CASRN 118-74-1)." *Environmental Protection Agency. Available Online: Http://www.epa.gov/iris/subst/0374.htm* .

Jardine, C. 2004. "Environmental Risk Assessment." Course Notes. University of Alberta.

Journal of Pesticide Reform. 1999. "Inert Ingredients in Pesticides: Are They Really Benign?" *Journal of Pesticide Reform.* 19(2):8.

Kaplan, Melissa. 2002. "Pyrethroids: Not as safe as you think." *Available Online: Http://www.anapsid.org/pyrethroids.html* .

Kensington Agriculture. 2004. Personal Communication.

Kern, M. & G. Vaagt. 1996. "Pesticide quality in developing countries." *Pesticide Outlook.* 7(5): 7-10.

Kimani, V.N. & M.A. Mwanthi. 1995. "Agrochemicals exposure and health implications in Githunguri location, Kenya." *East African Medical Journal.* 72(8): 531-535.

Kollman, W.S. 1995. "Summary of Assembly Bill 1807/3219. Pesticide Air Monitoring Results Conducted by the California Air Resources Board 1986 to 1995." Department of Pesticide Regulation. *Available Online: http://www.cdpr.ca.gov/docs/empm/pubs/ehapreps/eh9510ex.pdf* .

Krimsky, Sheldon (reviewer). 1997. "Biotechnology Safety: Report on Reports." *Environment (US).* 39(5): 27-30.

Leguizamon, E. 2001. "Transgenic plants in Argentina: present status and implications." *AgBiotechNet.* 3(ABN 077): 1-4.

MacDonald, Paul. 1997. "Cost of Production – PEI Table and Processing Potato Farms – 1994 & 1995 Crop Years." AGDEX #812/00. PEI Department of Agriculture and Forestry. *Available Online: http://www.gov.pe.ca/af/agweb/index.php3?number=71678&lang=E* .

Mackey, Maureen. 2003. "The Developing World Benefits from Plant Biotechnology." *Journal of Nutrition Education and Behaviour.* 35(4): 210-214.

MacPhail, Paul. 2003. Personal communication.

Majewski, Michael S. & David S. Baston. 2002. "Atmospheric Transport of Pesticides in the Sacramento, California, Metropolitan Area, 1996-1997." *Water Resources Investigations Report 02-4100.* Sacramento, California.

Matthews, H. Scott. 2001. "Analysis of the Benefits and Costs of Clean Air." In Fischbeck, Paul S. & Farrow, R. Scott (ed). *Improving Regulation: Cases in Environment, Health, and Safety.* RFF: Washington. 405-428.

Muhammad, I., M. Asrar & M. Ahmed. 1998. "General problems/constraints with private pesticide firms in the transfer of technology to the farmers." *Journal of Rural Development and Administration.* 30(3): 121-126.

NatureMark. 2004. *Available Online: Http://www.naturemark.com* .

NJDEP [New Jersey Department of Environmental Protection]. 1999. "WPS Fact Sheet: Carbamates." *Pesticide Control Program.* Trenton, NJ. *Available Online: Http://www.state.nj.us/dep/enforcement/pcp/bpc/wps/carbamates.pdf* .

O'Connell, Jim. 2002. "PEI's Perfect Potato: An Environmental Hazard?" *CTV W5.* October 25. *Transcript Available Online: Http://www.healthcoalition.ca/W5.pdf* .

Pray, C.E., J. Huang, R. Hu & S. Rozelle. 2002. "Five years of Bt cotton in China - the benefits continue." *Plant Journal.* 31(4): 423-430.

Prince Edward Island Potato Board. 2002. "Why PEI Potatoes?" *Available Online: Http://www.peipotato.org/why_pei.htm* .

Potato Blossom Industries Inc. 1998. "NatureMark Varieties." *Available Online: Http://www.potatoblossom.com/NatureMark.htm* .

Qaim, Martin & Zilberman, David. 2003. "Yield Effects of Genetically Modified Crops in Developing Countries." *Science.* 299(5608):900-902.

Reed, N.R. 1996a. In Pederson, T. 1997. "Dose-Response Assessment." *Available Online: Http://extoxnet.orst.edu/faqs/risk/dose.htm* .

Reed, N.R. 1996b. In Pederson, T. 1997. "Exposure Assessment." *Available Online: Http://extoxnet.orst.edu/faqs/risk/exp.htm* .

Reed, N.R. 1996c. In Pederson, T. 1997. "Hazard Identification." *Available Online: Http://extoxnet.orst.edu/faqs/risk/haz.htm* .

Reed, N.R. 1996d. In Pederson, T. 1997. "Risk Characterization." *Available Online: Http://extoxnet.orst.edu/faqs/risk/char.htm* .

Reigart, J.R. & J.R. Roberts. 1999. *Recognition and Management of Pesticide Poisonings, 5th Edition.* USEPA Document No. EPA 735-R-98-003. *Available Online: http://www.epa.gov/pesticides/safety/healthcare* .

Report of an Expert Panel to Review the Socio-Economic Models and Related Components Supporting the Development of Canada-Wide Standards for Particulate Matter and Ozone. 2001. S. Hrudey, chairman. Ottawa, ON: The Royal Society of Canada.

Reeves, Don. 2002. "2001 Pesticide Sales Report." *PEI Department of Agriculture and Forestry.* Charlottetown PEI.

Samy, M.M. 1998. "Constraints to effective transfer of corn technology as identified by farmers in Menoufia governorate, Egypt." *Alexandria Journal of Agricultural Research.* 43(1): 1-10.

Sivanarayana, G. 2000. "Adoption pattern and constraints in arecanut production technology." *Journal of Research ANGRAU,* Publ 2001. 28(4): 48-52.

Statistics Canada. 2003. "Preliminary Demographic Statistics October 2003." *Available Online: Http://www.statcan.ca/Daily/English/031218/d031218c.htm* .

Stratus Consulting. 2001. "AQVM Companion Manual for the Report of an Expert Panel to Review the Socio-Economic Models and Related Components Supporting the Development of Canada-Wide Standards for Particulate Matter and Ozone." Ottawa, ON: The Royal Society of Canada.

Szmedra, P. 1999. "Bittersweet harvest: the social costs of pesticide use in Fiji sugarcane production." *Journal of South Pacific Agriculture.* 6(2): 34-48.

The Economist. 2002. "Genetically modified crops: Seeds of Uncertainty". 362(8259): 70.

USEPA [United States Environmental Protection Agency]. 2004. "Evaluating Pesticides for Carcinogenic Potential." *Available Online:* *http://www.epa.gov/pesticides/health/cancerfs.htm* .

USEPA [United States Environmental Protection Agency]. 2004. *Available Online:* *Http://www.epa.gov* .

Viscusi, W. Kip & Joseph E. Aldy. 2002. "The Value of a Statistical Life: A Critical Review of Market Estimates Throughout the World," *Discussion Paper#392.* Harvard Law School: Cambridge.

WHO [World Health Organization] Recommended Classification of Pesticides by Hazard and Guidelines to Classification 2000-2002. International Programme on Chemical Safety. WHO/PCS/01.5. *Available Online:* *Http://www.inchem.org/documents/pds/pdsother/class.pdf* .

WWF [World Wildlife Fund]. Date Unknown. "Pesticides and Pesticide Management in Canada." *Available Online:* *Http://www.wwfcanada.org/satellite/prip/factsheets/PRIP_PesticidesManagament.pdf* .